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THE MODERN EXAMINATION AND TREATMENT OF PATIENTS WITH CHRONIC BRIGHT'S DISEASE

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(Read before the Arizona State Medical Association, at the thirty-eighth annual meeting, held at Prescott, April 18-20, 1929).

A vast amount of indeterminate research and a tendency both to hair-splitting and complex terminology and to multiple individual classifications, make it necessary to avoid, in an article of this kind, any extended discussion of the vast literature dealing with this subject.

Nephritis is the name applied to an inflammatory condition of the kidneys, and the kidney is different from all other secreting organs in the body both in development and in structure. The structural unit, the glomerulus, of which there are about two million, is beautifully adapted to serve as a single filter.

Since the description by Bright of the disease which bears his name, there have been numerous classifications. From the clinician and pathologist alike, the terminology has been confusing, as in one instance the diseased entity may be named from the structure involved, and in another from clinical findings. It appears, therefore that the old terms, parenchymatous and interstitial, with which there has been painful disagreement between the clinical and anatomical diagnoses, had best be discarded, and the classification of these nephropathies approached from the anatomical standpoint, which offers advantages for diagnosis, prognosis and treatment.

The following chart will give examples of this confusion, and under No. 1 will show how it may be clarified.

CHART

	No. 1 Essayist	No. 2 Addis	No. 3 Volhard & Fahr	No. 4 Frothingham
Acute Nephritis	(Glomerular (Tubular	(Hemorrhagic (Degenerative	(Nephritis (Nephrosis	(Glomerular (Tubular (including nephroses) (Combination of G. & T.
Chronic Nephritis	((Vascular (	(Arterio- (Sclerotic	(Nephro- (Sclerosis	(Final results of acute (Progressive vascular (Vascular (Vascular superimposed upon (healed acute nephritis

If we keep in mind classification No. 1, we will be able to talk the same language in consultation and it will not only guide in diagnosis, but will aid materially in the management. For example, if we find, following an acute illness, a patient edematous and urine loaded with casts, we will know at once that we are dealing with tubular nephritis and that we should restrict salt.

Attention is called to the rule that whatever type of pathology we have to start with usually predominates throughout the disease. However, due to close proximity of structure, there can hardly be one structure involved without some damage to the other.

In any condition where we have more than the norma lamount of albumen and casts, and if we have proof that it comes from both kidneys and is constant for a

time, to exclude transient albuminurias and cylindrurias found in forms of toxemias and bacterial infections, we may assume that we are dealing with some form of Bright's disease.

Dr. Addis, in a recent article, gave a resume of the findings in seventy-four apparently healthy medical students and showed an average of 1040 casts per twelve hours, and an average of 65,750 red blood cells per twelve hour. The white blood cells and epithelial were excreted at an average rate of 322,550 per twelve hours.

Picture in the mind's eye an uriniferous tubule and note what takes place in the different conditions. In the glomerular, at the site of lesion there is proliferation of endothelial cells lining the capillaries in the glomerular tufts, proliferation of the cells lining the capsular spaces, exudation of serum into the capsular spaces with blood cells or leucocytes, and thrombosis of some glomerular vessels.

In the tubular, we find swelling of the epithelial cells in the convoluted tubules and, to a lesser degree, the loops of Henle, with abnormal amount of fat and, in some instances, the appearance of hyaline-like material in these cells.

Differential Diagnosis

	Glomerular	Tubular
1. Color of urine	Dark	Light
2. Red blood cells	Large amt.	Few
3. Retention of non protein nitrogen in blood	Marked	None
4. Blood pressure	Elevated	No elevation
5. Casts	Few	Many
6. Edema	Slight	Marked
7. Retention of chloride in blood	None	Marked

In chronic nephritis, one or another of these may dominate the clinical picture.

Vascular nephritis is a progressive degenerative process in the small afferent arteries and the vessels of the glomerular tufts, and due to process of repair there is formation of scar tissue, and the end result is a small contracted kidney.

In vascular nephritis the symptoms are usually insidious and from five to fifteen years may pass before symptoms will be such as to call the attention of the patient. As a rule, the circulatory system will be first to attract attention, hypertrophy of the heart, elevation of blood pressure, the arteries may become palpable and tortuous. Headache is a common symptom, shortness of breath, loss of weight, digestive disturbance and impaired vison.

Nocturia, with increased output at night over day, is a later sympton. At first the urine may be normal, then small amount of albumen appears, and then casts and red blood cells. As the disease progresses the specific gravity becomes stabilized as the kidney lacks the power to vary the concentration. Edema will accompany beginning myocardial failure.

In determining the excretion of solids, the specific gravity should be noted over a long period of time, as so many factors go to make up the total.

What is known as the "water test" is an excellent way to determine the functioning power of the kidney. Have the patient drink 1500 c.c. at 8 a. m., on fasting stomach, and take no food or other fluid before noon. The specific gravity may vary from 1002 to 1025 in a normal individual, but in disease it depends on the degree of destruction present, and may not vary more than two or three points.

EXAMPLE
WATER TEST

	Case 1, Healthy		Case 2, Sick	
Sp. gr.	Quantity	Sp. gr.	Time	Quantity
8:00 a. m.	180	1010	30	1016
8:30 "	360	1003	—	—
9:00 "	390	1002	40	1018
9:30 "	255	1004	—	—
10:00 "	160	1010	35	1016
11:00 "	100	1011	—	—
12:00 "	80	1011	85	1016
	1525	1005	190	1016

The phenolsulphonephthalein test is the most reliable, accurate and easy to carry out. Fifty per cent excreted in two hours and ten minutes is considered normal. As disease progresses to terminal stage, none will be excreted in the first two hours.

Examination for nitrogen and chlorides should be made, as they aid in outlining treatment and, as disease progresses, chlorides and urea decrease to minimum. One half of all solids are urea and normally there is excreted 20 to 35 gm. in twenty-four hours. The amount of chlorides is 10 to 15 gm. in twenty-four hours.

The degree of nitrogen retention in the blood has come to be relied upon as an index of the functional efficiency of the kidneys.

It is customary to divide the nitrogen-containing constituents of the blood into two groups, one the proteins (albumens and globulins) and the other the various non-protein nitrogenous substances (utilized food derivatives, waste matabolic products, etc.). Because of the information on the problems of metabolism and excretion, the non-protein group, and particularly urea, uric acid and creatinin, are made.

According to Myers and Fine, there is a definite order in which these substances are retained and uric acid shows first. As the disease progresses, urea also begins to accumulate; next creatinin, and the latter is

the surest guide to a prognosis, and a rise to 5 mg. warrants a hopeless prognosis.

When we know of the similarity of the pathology in the afferent vessels in the kidney in vascular nephritis and in the vessels in generalized arteriosclerosis, and knowing that many cases of arteriosclerosis have their primary lesion in the kidneys, may we not assume that the cause is the same in vascular nephritis, and arteriosclerosis?

Because there is no specific treatment for chronic nephritis, we should not sit idly by, but should be on our guard to give advice that may prevent the establishing of the malady, or at least to modify its ravages.

Doctor Harriman starts his discussion of management by saying, "If possible, remove the cause. Let the patient be moderate in all things."

Let us continuously bear in mind that all cases of arteriosclerosis and hypertension are potentially cases of chronic nephritis. Then it is our duty to warn our patients to conserve their future health by guarding against alcoholics, the abuse of their vital organs by the over-use of tobacco, all foci of infection especially teeth and tonsils, over-eating, constipation, overwork, and (possibly the worst offender in carcio-vascular-renal disease) worry.

Allen states that all cases of hypertension can be cured by salt-free diet. This, of course, is a broad statement but undoubtedly has at least some merit and, realizing the similarity of pathology and the hand-in-hand method of vascular-renal disease, why should we not at least advise all questionable and suspected beginning cases to be moderate in the use of salt?

Doctor Blumgarten discusses the rational treatment of chronic nephritis from the view-point of functional pathology, but, at the same time, calls our attention to the fact that function depends invariably upon structure. While we cannot determine the amount of renal functioning tissue in any given case, we CAN determine the renal functional capacity, the kidney's ability to excrete the different constituents of the urine and whether the dominant lesion is tubular or glomerular.

In instances in which the water output is persistently low, in which the main lesion is evidently a glomerular one, treatment holds out but little hope.

We not only can determine the chief lesion, but also can determine the difference between the instances with, and the instances without, retention of waste products.

We should try to attack the lesion itself more actively instead of adapting the treatment to the inevitable outcome of the affection.

Blumgarten advises decapsulation as a rational operation and one that should be given a trial more frequently in early instances of this disease—particularly in those cases showing repeated acute exacerbations.

In instances minus retention of waste products, pay greater attention to regulating the general regimen of the patient's life than to restricting his diet or treating him with medicines. The total amount of food ingested should be limited; at the same time, the proteins should be restricted very little, if at all. It is more important that the patient's nutrition be maintained than that a scientific balance of elimination be reached.

In instances with retention of protein waste products, eliminate such retention as much as possible. We may accomplish this by dietetic means—by decreasing the protein intake without sacrificing caloric requirements. With uremia absent, or not imminent, the protein tolerance may be marked without producing retention of waste products in the bloodstream. To secure this tolerance, we commence with starvation, then increase the proteins (by degrees) until tolerance is established.

We ought to anticipate acidosis by the employment of alkalies. When this trouble does arise, intravenous injection of alkalies will be found the most effective way of treating it.

Venesection should be done where we suspect a beginning uremic condition.

Edema can usually be kept to negligible amount in the tubular type by restriction of salt, but when it reaches proportions where some drastic means have to be taken, we have found ammonium chloride given for twenty-four hours and followed by novasurol has netted, in a number of cases, more than one gallon of urine in the twenty-four hours following.

Such results are very flattering, but only prolong the agony for days or weeks, whereas the preventive treatment may prolong life for years.

SUMMARY

Simplification of classification, which aids in diagnosis and treatment.

Normal urine may contain many pus cells, casts and blood.

How to differentiate between glomerular and tubular.

Chemical analysis of blood is guide as to progress of vascular nephritis.

Similarity of pathology in vascular nephritis and arterio-sclerosis.

That chronic vascular nephritis may be slow, but is a progressive disease and preventive treatment should be instituted early as in advanced cases the treatment is only symptomatic.

URINARY FINDINGS SIMULATING NEPHRITIS

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(Read before the Arizona State Medical Association, at the thirty-eighth annual meeting, held at Prescott, April 18-20, 1929.)

This paper is intended to be of some aid to those doctors in general practice, or some other special line of endeavor, where a routine urine report showing the presence of albumen frequently causes them to make a diagnosis of Bright's disease without inquiring further into the kidney condition. We must bear in mind that inflammation along the urinary tract causes the kidney and mucous membranes of the tract to pour out mucous or serum albumen which will throw down a cloud when the ordinary tests for albumen are applied to the urine.

A urinalysis is not complete until a microscopic examination has been performed.

Kidney inflammation causes a hyaline drop formation in the renal epithelial cells, with extrusion into the renal tubules, where casts are formed. These are in turn forced out in the urine[1]. This does not necessarily mean Bright's disease. Albumen and casts are often found in varying amounts in bacterial infections along the urinary tract and are associated with pus and blood. In fact, pus or blood either, will show a marked presence of albumen on chemical analysis.

This paper will deal with the presence in the urine of pus and blood and will not mention albumen and casts from now on, hoping that we can clear up the fallacy in the minds of many concerning the value of chemical analyses alone.

In obtaining urine from a female, the finding of pus microscopically in a voided specimen is valueless. The vulva should be thoroughly cleansed, and the patient catheterized. The constant presence on the vulva of a leucorrheal discharge always contaminates voided urine. When obtained in this manner we ordinarily disregard the finding of an occasional red blood cell, its presence being due to trauma.

Repeated studies have shown that one finds five to seven leucocytes to the high power field, in the urine of normal individuals[2]. Unfortunately the presence of blood in the urine is more apt to attract the attention of both the patient and the physician than a turbidity, unless it be very marked.

Whenever one finds pus in the urine, a most careful history must be taken, with especial reference to gonococcal urethritis; repeated chills and fever; pain, whether localized or radiating; dysuria, whether initiative or terminal; frequency, etc.

In the female, the local urethritis of gonorrheal origin is usually of brief duration and of moderate severity, the infection preferring to involve the genital tract. The anatomy of the female urethra, with the relaxation occurring after childbirth, renders this tract prone to subacute and chronic infections.

In the male, we should search for evidence of persistence of infection, originally gonococcus, but later superseded by ordinary pus-forming organisms, or a primary non-specific urethritis; the presence of strictures, in either the anterior or posterior urethra, cowperitis, infections of the glands of Littre, or the lacunae, para-urethral abscess, etc. The most common and persistent cause is chronic prostatitis and vesiculitis.

The acute infections are frequently accompanied by dysuria[3], very often without general systemic disturbance as long as the process remains localized in the urethra or bladder. With extension to, or acute involvement of, prostate or epididymes or kidney, the picture becomes dramatically altered and the patient manifests systemic reaction with fever, chills and general malaise in addition to localizing features.

In considering the bladder sources of pus one must bear in mind that this organ is practically never involved primarily, but secondarily, from extension from above or below. However, there are certain intrinsic pathological entities encountered here that invite and aggravate these secondary invasions. Bladder neoplasms or tumors are not usually painful until this secondary invasion takes place.

Chronic urinary obstruction at the bladder neck from prostatic hypertrophy, fibrous contraction, median bar obstruction, congenital valve formation, functional sphincter disturbances, certain cord lesions, all result in urinary stasis and secondary infection with pus formation. We get a loss of bladder tone with subsequent dilatation, infection, saccule formation from protrusion of vesical mucous membrane through overtaxed muscle bundles and stagnant infected urine; from which sediment, calculous formation occurs.

False diverticuli are formed as the result of obstruction and true diverticuli then make their appearance. A true diverticulum is an outpouching of the bladder wall, including all its intrinsic layers and connected with it by a narrow neck. Its capacity may vary from half an ounce to that equal to the bladder itself. A true diverticulum is probably congenital in origin, but, owing to its anatomy, it is very prone

to infection, especially if there happens to be obstruction of the bladder neck, when it is most resistant to ordinary methods of treatment.

In prostatic hypertrophy the systemic disturbance depends upon urinary obstruction and secondary infection. Residual urine may remain clean for years, but such is infrequently the case. With the advent of infection involving bladder, ureter and kidney, the march is quiet and insidious, accomplishing a gradual destruction of functional kidney value of which the patient remains relatively unaware. As far as symptomatology is concerned, aside from perineal pain, which is by no means constant, there is nothing to suggest malignancy. Certainly no evidence is afforded from urine analysis. Palpation of the gland by rectum may reveal important evidence. Pathological examination finds evidence of malignancy in one of every five prostates removed surgically.

Other common sources of primary vesical pyuria are granulomas, granular and similar types of chronic cystitis, tuberculosis, and communication with perivesical foci of infection, such as rupture of appendiceal, tubo-ovarian and similar abscesses into the bladder.

Cystoscopy alone, or urethro-cystoscopy, aided by plain radiography and cystography, will enable one to localize a pyuria as being of vesical origin, far more rapidly and accurately than any other method.

Of the pathological conditions of the ureter responsible for pyuria, we wish to call attention to strictures of pre- or postnatal origin, calculi or neoplasms, non-tuberculous infections and tuberculosis—all of which come strictly in the field of the urologist.

At the kidney level, the most common infection to be considered is the non-tuberculous pyelonephritis. In etiology, as elsewhere in the urinary tract, obstruction is the important provocative feature. All forms of mechanical obstruction below the renal pelvis will favor the incidence of infection. Such obstruction may be at the bladder neck by prostatic hypertrophy or median bar formation, or within the urethra from stricture. It may be along the course of the ureter from internal obstruction or from external pressure by an enlarged uterus, or from another pelvic or intra-abdominal tumor. Or it may be a kink or twist in the course of the ureter due to anomalous position of the kidney, or from an anomalous blood vessel supplying the kidney itself. Such conditions favor urinary stasis and, indirectly, infection. With the development of infection, stone formation is next in order. It has recently been shown that infection always precedes stone formation.

The development of secondary pyelitis from a remote focus of infection is well recognized, such secondary infection occurring in the course of acute infections, like otitis media, pneumonia, scarlet fever, typhoid fever, and in more prolonged infections, such as chronic middle ear disease, chronic osteomyelitis or chronic empyema. We must not overlook the possibility of these secondary infections becoming permanent. Frequent cases have been noted where the patient recovers from the original acute infection only to fall a victim years later to the end results of the untreated renal infection which had been either ignored or not recognized.

The combined presence of stone in the kidney pelvis with attendant infection, accomplishes a diminution of that organ's functional value. If the attendant infection be slight and drainage free, renal degeneration may be very slow. If there be recurrent attacks of pelvic retention with attacks of subacute pyelonephritis, the damage becomes more rapidly accomplished. Chronic renal infection and stone may reduce the kidney to a flimsy pus sac possessing no value as a functional organ. With free drainage, constitutional symptoms may be lacking. The urine may be constantly clouded with pus or intermittently so, as the renal sac empties itself from time to time. In such cases the bladder and urethra are tolerant and the only local subjective symptom may be slightly increased frequency. The urine will be turbid throughout, even after thorough bladder lavage.

The tuberculous renal infection manifests itself first by nothing more than increased frequency, the urine, thus early showing only a slight trace of albumen. The tubercle bacillus may be demonstrated by examination of urinary sediment or, more likely, after guinea pig inoculation. The pathology at such early stages occurs in the cortical portion of the kidney or near the tip of the renal pyramid. As soon as a tubercle ulcerates through to the pelvis the amount of pus and blood is increased, though such may occur long after the development of annoying urinary symptoms. The first symptom may be hematuria occurring as the result of early nephritis. As soon as the bladder becomes involved, dysuria becomes more intense and urgency and frequency make life nearly intolerable. The acid urine does not cloud on standing and, if cultured, usually gives no growth. Secondary infection supervenes late, if at all. The demonstration of the tubercle bacillus by staining methods, or the recovery of

that organism from the inoculated guinea pig, establishes the diagnosis.

HEMATURIA

Blood originating in the urethra results ordinarily from abrasions through the mucous lining from external or internal traumatism. Acute urethritis and urethral caruncle may cause primary bleeding.

At the bladder neck, in either sex, the multiple benign polypi may cause bleeding.

In prostatism, with increasing congestion, which at times becomes considerable in the median lobe of the gland, a degree of hemorrhage occurs which may assume serious proportions. This is the bleeding often giving rise to an ill-founded fear of malignancy.

Simple acute ulcer of the bladder causes bleeding, frequently attended by intense dysuria and vesical tenesmus. These excoriations are usually located in the bladder wall. One particular group, situated on the anterior wall above the internal meatus, comprise the so-called elusive ulcer of Hunner.

Acute cystitis and acute exacerbation of a chronic cystitis are frequently accompanied by a terminal bleeding and strangury. Tuberculous involvement of bladder and ureter frequently cause bleeding.

New growths originating in the bladder seldom give warning of their presence prior to the tell-tale painless hematuria.

Stone in the bladder may announce its presence by visible bleeding. It is the attendant infection or underlying cause, however, that sends the patient to his physician.

Injuries to the bladder and ureter are mentioned only in passing.

Excoriations from the passing of ureteral calculi cause bleeding. According to Hunner, ureteral stricture is a very important cause. The occurrence of hematuria in association with appendicitis has been reported on several occasions. A confusing problem for diagnosis thus presents itself and every resource of diagnostic procedure and surgical judgment must be enlisted.

At the level of the kidney, stone is the most common factor; however, the larger calculi are usually silent. Acute pyelitis and pyelonephritis, as a rule, produce a urine in which red cells occur as commonly as does pus. Tuberculous pyelitis frequently announces itself at an early stage by hematuria, commonly painless. Like most bleeding of renal origin, the urine as voided is smoothly admixed with blood and appears as a homogeneous wine colored solution.

Neoplasms of the kidney usually remain unsuspected prior to bleeding, which is characteristically painless.

Idiopathic bleeding from the kidney or essential hematuria, in our opinion, is a misnomer and never occurs, for there is always a cause. It may be impossible to find it, however.

All cases showing pus and blood in the urine should have a culture made on an aseptically collected specimen, where the presence of a growth will indicate that the condition is not a Bright's.

Albumen, pus and blood, without the other classical signs of Bright's disease, such as an increase in blood pressure, in blood nitrogen and blood chloride, edema, and general toxic symptoms, should be subjected to a routine urological study by a comeptent urologist.

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DISCUSSION

DR. FRANK J. MILLOY (Phoenix): I have listened to the papers of Doctors Davis and Littlefield with much interest. They have said a great deal in the time allotted. There is little controversy in the things they have said, so all that remains is to emphasize some of their statements and possibly add a word or two. As Dr. Davis has said, there is no classification which covers chronic nephritis, because one type always overlaps another. However, from a purely clinical standpoint, we might make two divisions; namely, nephrosis and nephritis. Nephrosis may include the tubular form. The pathology of nephrosis is a hyalin degeneration of the uria and cholesterinemia. There is no cnange in nria and cholesterinemia. There is no change in blood chemistry nor in blood pressure. This probably represents by far the minority of cases.

But the clinical picture we have of members of that great multitude who are afflicted with Bright's disease, includes the condition of the kidneys, the heart and the arteries. These organs are inseparable and the diagnosis requires a thorough investigation of all three. The most valuable information about the urine is obtained from the Mosenthal. It will show a fixation of specific gravity, the inability of the kidney to concentrate, and the night output equal to or exceeding the day output in quantity of fluid, chlorides and nitrogen. This is chronic nephritis. The prognosis from this point depends upon the rate at which damage develops in the arteries and the myocardium. As Dr. Davis has said, chronic nephritis is vascular degeneration or change in the glomerular system. This vascular change extends to involve the whole arterial system. The blood pressure rises. The ophthalmologist can often give us the best clue as to prognosis. Eye-grounds with tortuous, edematous arteries and retinal hemorrhange spell disaster.

If we are not dealing with the advanced malignant type of hypertension, the salt-free diet, as advocated by Allen, very often produces wonderful results. The salt-free diet means more than instructing patients to eliminate salt from their food. It means the reduction of the chlorides in the unrine

to 1½ grams daily. This is extremely difficult. I have found it almost impossible to get it below 3 grams in 24 hours. The fluid intake must be reduced also to one liter daily. This procedure is of wonderful value in reducing excessive weight also, which is most essential. Evidence of failing myocardium is a sign of danger. Edema is often present. Here, again, limit salt and water intake and use digitalis. This does not mean 15 drops of digitalis three times a day. There are a number of formulae, but, roughly speaking, one ounce of the Parke-Davis tincture will digitalize a patient. This should be given in three to five days, depending on the severity of the decompensation. The electrocardiograph is of extreme importance in watching the exact conditions of the heart. It will indicate the point of digitalization and then a maintenance dosage should be given. If edema is present, diuretics should be given:: ammonium chlorid, novasural, theocin, etc. If these fail at first, it is marvelous how they will work sometimes after the heart is digitalized.

Another way of viewing the prognosis of chronic nephritis is to consider the usual termination. Marked increase in blood chemistry means the advent of impending coma, unless, of course, we are dealing with an hypertrophied prostate. In addition to the above procedure, glucose intravenously is of extreme benefit. But few patients reach this stage. The commonest terminations are pulmonary complications and vascular accidents. The chronic nephritic has little chance with pneumonia. The vascular accidents include cerebral hemorrhage, coronary thrombosis, and the various forms of emboli. It is not an uncommon thing for a desperately sick patient, who had a marked edema and advanced decompensation, to be brought back to the point where he is compensated and free from edema and apparently well, and then suddenly to drop dead. This means a vascular accident, generally a coronary thrombosis or cardiac infarct.

One more point in prognosis. Chronic nephritis occurs in families and it is a common occurrence for whole families to die at about the same age, usually the early sixties. As Barker says, "If you would avoid high blood pressure, select your ancestors."

DR. R. J. STROUD (Tempe): Dr. Rountree first expounded the use of ammonium chlorid in failing compensation of the heart and had good results. Dr. Allen, while enthusiastic about salt-free diet, pointed out that he never used ammonium chlorid in cases because that must first make an acidosis. He has made that very distinct statement twice.

DR. ROBERT FERGUSON (Bisbee): I should like to report one case, a man of fifty, apparently well with no previous history; went to bed at night and was found in the morning with convusions and paralysis, pupils dilated during convulsions, pressure 120/80, no edema in the lungs or anywhere. He was unconscious and therefore I did not get a history. The urine had a large amount of albumin casts. The patient died within twenty-four hours. I made a diagnosis ofuremia.

DR. W. W. WILKINSON (Phoenix): A little personal experience during the last year in the direction of the amount of water used individually. For several years I noticed cold showers every morning gave me a little rheumatism, and I could not eat citrus fruit. Dr. Percy, of Los Angeles, last year spoke of the amount of water which should be used. He stated that sixteen glasses should be taken every morning, that is about four quarts, which seems rather excessive, but for the past few years I have used two quarts a day and that seems not excessive for a man of two hundred pounds. I should like the Doctor to tell me if one can drink too much water?

DR. D. F. HARBRIDGE (Phoenix): I was rather pleased to hear, in Dr. Milloy's discussion, the use of the opthamologist in this sort of lesion. The purely clinical investigations and diagnoses based on results of personal experience, knowledge and sometimes intution, have in recent times been overshadowed by laboratory methods. Medicine is still an art and not a science. The body of your patient is unquestionably the best laboratory in the world. Dr. Milloy referred to certain lesions which are found in the eye-ground in well-advanced, frank cases, but it takes a big intuition, a big experience, to be able to examine the fundi of patients and determine the possibility of a future development of some kidney lesion. It is usually enough to say that the vessels show arteriosclerosis and that there are patches of exudate and hemorrhage. These are the frank end results, but if you wish to protect your patient properly, when you suspect any possibility of a vascular kidney lesion, the wisest thing is to have the eye-ground examined and examined carefully. This is not a perfunctory thing but it takes time and repeated visits to fully interpret the findings. In many of these lesions, some of the very earliest findings involve the fine blood-vessel twigs, and I know of no other place in the human body where the blood-vessel can be studied so clearly and accurately as in the fundus of the eye (by the eye man). There are certain definite changes which occur some time before more frank lesions are found. To illustrate: An official of one of the mining camps came to me. I examined the eye-grounds and found these certain little changes which indicated, to my mind, that the man was on the danger line. I inquired into his history and found that, while he used alcohol to a certain extent, not excessive, he was a large, irregular eater. I cautioned him in this regard and he readjusted his life. For three years he went along and urine examinations showed no albumin. He broke loose again, and came back to me. Further examination of the eye-ground showed more progessive difficulty and still the urine showed no findings of value. He was cautioned again and lived for two years in a restricted way. He broke loose again and went along for a year or two, and then suddenly developed a severe acute nephritis and died. This is simply one experience of a good many. Had he accepted the original advice and lived accordingly, I think he would have been alive today. All these changes precede those found by laboratory methods and it is exceedingly important to recognize them.

DR. DAVIS (closing): I have been trying to clear up for myself that word "nephrosis", a word coined in 1925. It is a confusing term. Just what does nephrosis mean and what does it include? I think if wewould stay with the pathological classifcation we would be better off.

Ammonium chlorid, in my practice, is not given other than as a diuretic for twenty-four hours preceeding novarsol.

As far as water is concerned, I do not think anybody ever drinks too much water unless suffering from some form of Bright's disease.

In my paper I mentioned the eye, difficulty of vision, palpable edema and retinal hemorrhages.

One point I laid stress on was worry. To show that that holds true: After I finished writing this paper I took my blood pressure, which has never been above 130, and found it to be 160.

DR. LITTLEFIELD (closing): The principal thing I tried to do was to get the general practitioner away from the idea of a urinalysis for albumin and sugar as a complete examination.

THE SURGICAL KNEE

JOSEPH MADISON GREER, M. D.
Phoenix, Arizona

(Read before the annual meeting of the Arizona State Medical Association, held in Prescott, Ariz., April, 1929.)

From time to time there has been considerable written and much discussion relative to the "Surgical Abdomen", in which various conditions of the abdomen that are properly diagnosed or treated by surgical means are considered. Therefore, it occurred to me that it might not be totally uninteresting to consider for a few moments some of the conditions of the Knee-Joint that probably should be diagnosed or treated by surgical means.

In making comparison betwen the conditions of the abdomen and conditions of the knee-joint, that require surgery, you may advance the criticism that this is quite farfetched, and that we have nothing in the knee that corresponds to the acute surgical abdomen, in which immediate surgical intervention is demanded. I am not so sure that this would be true and cite, for example, the acute injuries of the knee in which there is hemorrhage into the joint. This, to the minds of some of us, demands early surgical treatment to remove the extravasated blood. It is true that delay here may not endanger the life of the individual, as it would with a surgical abdomen, but it may endanger the integrity of the knee-joint and prolong its disability. In further support of the analogy between the knee and the abdomen, may I say that Dr. John Moorhead, of New York, one of our foremost traumatic surgeons, frequently speaks of "The Laparotomy of the Knee Joint".

In this short paper I shall not present anything original, nor shall I cite any large series of cases. However, I do feel that, by calling attention to some of the more common injuries and conditions of the knee-joint that seem at this time to be properly treated by surgery, and by encouraging ourselves to a more careful consideration of surgical treatment, after thorough examination and effort at diagnosis, many of our present knee-joint disabilities may be eliminated and many others shortened. We have ever attacked the knee-joint by surgical means with fear and trembling; and rightly so, for an accident in the way of infection of the knee-joint following surgical intervention is a serious thing and tends to promote modesty and humbleness in the person of the surgeon. It has almost the same effect upon the feelings of the surgeon that the plucking of the tail feathers of a strutting peacock does upon the fowl. He would just a little rather not be seen for awhile. However, of late years, with our modern methods of surgery, we do not fear this so much and we do not hesitate to open wide the cavity of the knee-joint and make a thorough inspection and exploration.

Now, let us say a few words about the structure of the Knee-Joint. As you know, the knee-joint is not a simple hinge joint. It has three articulations: The condyles of the femur with the tuberosities of the tibia, and the femur with the patella. Gray states that, in certain animals, there are three synovial sacs, and developmentally this is true in the human and these three sacs fuse, forming one large one, the largest in the body. This large sac is lined with synovial membrane and has a row of villous-like processes extending into it.

The semilunar cartilages are rather sickle-shaped with broad convex margin attached to the joint capsule and a narrow concave margin extending free into the joint cavity. These cartilages lie between the bones, though actually outside the synovial sac. They are quite loosely attached to the toe of the tibia and, when the joint is moved, there is a slight movement of the cartilages on the head of the tibia. So you see that this mechanical arrangement and relatively insecure attachment has something to do with the conditions we find following injury of the knee-joint.

Another thing which we should keep in mind relative to the structure of the knee-joint is the rich blood supply of the synovial membrane and the fact that this blood supply gets its origin from branches which penetrate the joint at the attachment of the lateral ligaments. The articular cartilage normally derives most of its blood supply from osseous branches which penetrate the head of the bone and supply the deeper growing layer of cartilage. The surface layer of the articular cartilage is avascular. The wall of the joint consists of the fibrous capsule and the synovial membrane. The synovial membrane lines the entire cavity, including the margins of the articular cartilages, where it acts as a perichondrium. It is lacking over the weight bearing areas, which are simple avascular cartilages. Remember that this is the largest synovial sac in the body and may contain thirty-five to fifty cubic centimeters of fluid without detection and may hold several times this amount.

One of the striking features of the connective tissue which forms the important supporting substance, is the presence of mucin-producing cells which are present in considerable numbers and produce the viscous element of the synovial fluid, this se-

cretion being expressed by the normal pressure during normal movement of the joint.

Another somewhat interesting thing about the structure of the joint from the standpoint of injury and internal derangements, is that this connective tissue supporting the secreting layer is supplied, at all points where packing is required, by masses of adipose tissue. The infrapatellar fat pad is an example. Some most interesting and important things relative to the physiology of these structures are pointed out by Fisher, in his most excellent monograph, "Internal Derangements of the Knee Joint": First, that the synovial membrane is capable of absorbing fluids of various types, whether true solutions, colloids or suspenoids, BUT with VARYING facility in the different types; second, that the presence of a foreign body in a joint causes a synocial reaction, resulting eventually in its attachment to the villi, after which it is enveloped by newly formed connective tissue; this explains one type of foreign body, or joint mice.

After this very brief and incomplete discussion of the structure and function of the joint, let us consider some of the surgical conditions that we see most frequently.

First, let us discuss the acute injuries with fluid in the synovial sac. It is important for us to realize that, in an acute knee injury which presents pain, tenderness and marked swelling within a few hours after the injury, whether we can get an accurate sense of fluctuation or not, although we usually can, there is hemorrhage into the synovial sac. We all know that a fairly constant finding when we open up a chronic knee-joint, with a history of injury, is the presence of much old blood pigment in granular form throughout the interstitial tissues of the hypertrophied synovial membrane. This is evidence of an attempt, and many times an unsuccessful attempt, at absorption of hemorrhage that occurred at the time of the original injury. We also find other chronic inflammatory changes with hypertrophy and hyperplasia, which are brought about, no doubt, by the presence of this blood tissue in the joint, which, even though it is a fluid tissue, acts as a foreign body and tends to produce adhesions and other troubles. Therefore, given an acute injury of the knee with pain, swelling and evidence of fluid in the joint, without trying to make an accurate diagnosis as to whether it is an injured semilunar, a lateral ligament or what not, let us call it an "Acute Surgical Knee" and immediately apply surgical treatment as follows:

Have the patient lie on a table with the leg and thigh extended over the edge to a point above the mid thigh and the heel resting upon a support, this so that the surgeon may have easy access to the leg, knee and thigh. Then carefully, under aseptic precautions (and these can be carried out adequately in the surgeon's office), aspirate the blood from the joint with a large caliber needle, going upward and inward from a point corresponding to about nine o'clock for the right knee and three o'clock for the left knee, in relation to the patella. One will frequently be surprised at the amount of blood that can be aspirated. This procedure may be facilitated and made more complete by the application of an elastic bandage and also by the injection of ether, although the latter is somewhat painful for a few minutes. When all the fluid possible is removed, a closely-fitting, well-padded plaster cast is applied from the junction of the lower and middle third of the leg to the middle of the thigh and the leg is kept elevated at an angle of about forty-five degrees. This degree of elevation is maintained constantly for from forty-eight to seventy-two hours. Then the leg may be brought down gradually but elevated again promptly at the slightest suggestion of swelling. This cast is kept on, without being disturbed, for a period of at least two weeks. Then mild active motion without weight-bearing is commenced. This is continued, with weight-bearing, and so on to recovery. Physiotherapy may be used if indicated.

The things accomplished in this treatment are the following: By removing the extravasated blood, which is a tissue and acts as a foreign body, we take the burden of absorption from the tissues and leave them more free to aid in repair. We remove the pressure of the extravasated fluid and allow a better circulation. By complete and thorough immobilization and elevation, we prevent further injury and further extravasation of blood and also further pressure, which interfers with the active blood supply. By maintaining this for a period of about two weeks, if there is injury to the cartilage, the physiology of repair will commence about this time (according to Sir Robert Jones) and will be continued under the stimulus of moderate active movement from then on. May I just mention, in passing, that three cases have been treated with this method during the last three months and all have been normal within five or six weeks' time. By this I mean entirely normal, with no pain, swelling or stiffness.

A few of the chronic knee-joint troubles should be mentioned, as they are surgical problems. I shall not dwell upon these at any great length as our instructions were to write our papers in such a way that they might be of value to the men in general

work and it is not expected that men doing general work would care to take the time and trouble to attack surgically chronic knee-joint troubles.

Among the more common chronic affections of the knee-joint that are surgical and that we should be familiar with are chronic or recurrent dislocations of the semilunar cartilages, usually the internal, and simulating lesions. I put it this way because there are other lesions of the internal derangements of the knee-joint that cannot be distinguished from dislocated cartilages and that is the reason we advocate wide incision and thorough exploration in such conditions, as I shall mention later. In an analysis of symptoms of these chronic dislocated cartilages, as outlined in a report by Dr. J. O. Wallace, of Pittsburg, in his excellent article published in the "Journal of Bone and Joint Surgery", on "Internal Derangements of the Knee-Joint", he gives the following in a series of seventy-one coses:

1. History of injury: eigthy-nine per cent.

II. Pain and tenderness: almost one hundred per cent and the sites of pain and tenderness vary considerably.

III. Swelling: ninety-three per cent.

IV. Weakness: ninety per cent.

V. Locking: seventy-five per cent (you see they do not all have a history of locking).

VI. Lack of complete extension: about one hundred per cent.

X-ray findings: usually negative. This is a very important thing for it seems to me that at times we are misled when we get a report of negative x-ray findings.

Other affections are: joint mice, villous synovitis, etc., which I shall not dwell upon here. The treatment which is recommended in these cases is, briefly, as follows: Wide incision and thorough exploration with adequate surgical procedure depending upon the condition found. This should, preferably, be a long mesial patellar incision with a rotating of the patella outward, done under the Lane, or non-touch, technic. Some men use the split patella incision and are not so particular about non-touch technic. For after treatment, a moderate period of immobilization to allow adequate repair seems reasonable. However, I am aware of the fact that some surgeons start movement almost immediately, with seeming good results.

CONCLUSIONS

1. There is such a thing as an "Acute Surgical Knee", demanding early and prompt surgical treatment to prevent damage and alteration of the knee-joint structures, which, when once established, require a long time to correct.

2. This prompt surgical treatment may be carried out in the hospital, by the general practitioner, or even in his office.

3. If this treatment is carried out promptly, many of the chronic knee-joint troubles will be eliminated.

4. Many of the chronic knee-joint affections are surgical problems and can be corrected by wide incision and thorough exploration and, may I again say, in the words of Dr. John Moorhead, by "Laparotomy of the Knee-Joint".

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DISCUSSION

DR. A. C. CARLSON, (Jerome): I first wish to compliment the doctor on a very good paper on an interesting subject, especially of interest to the men in the state who are doing industrial surgery.

A few points of interest I agree with and some I disagree with. Early aspiration should be emphasized very strongly, especially in cases where the blood soon thickens and it becomes very difficult to aspirate the coagulum through a moderate sized needle. Another point of interest is the point of entrance of the needle. The doctor says he enters from the outside; you can enter an inch from inside and get the same results with no danger. A point the doctor brought out, that I do not agree with, is immobilization after aspiration. There is very apt to be recurrence of effusion, necessitating repeated aspirations, and if the knee is confined to a cast, the occurence of an ounce to an ounce and a half of fluid may not be detected. Many authorities advise us to put on no splinting, to use active motion but not passive motion and no physiotherapy. The knee can be supported by strapping or a lateral hinge support and still permit action of the knee. No patient will traumatize further because he is not going to move this knee to the point of pain.

DR. R. D. KENNEDY, (Globe): We men in general practice should be conservative in our treatment of injuries to the knee joint. There is one injury the doctor did not lay much stress on,—rupture of the internal lateral ligament. We are going to see more of this as time goes on and the number of automobiles multiplies. In the large cities they are seeing quite a few of these cases in which the patient is struck on the outer side of the knee and the internal lateral ligament is torn. There is enormous swelling and you might have difficulty in diagnosing rupture of this ligament, but if you will just repeat the process of the injury you will find you are able to put your finger right down into the joint where torn. If this condition exists it is best to go into it right away,—the internal semilunar ligament should be repaired; if not, scar tissue is weak and will leave a weak joint.

Another common cause of swelling in the joint is pinching of the post patellar fat pad. If suspicious of that, a high heeled shoe should be worn so that the knee is bent and does not bring full extension. We seldom see this condition in women because they seldom straighten the knee.

In regard to dislocation of the semilunar cartilage: if once you get the knee straight it is reduced, and if immobilized for two weeks, the chances are it will stay in place. If there is a history of two or more dislocations of that semilunar cartilage, permanent reduction can never be obtained; scar tissue is too dense.

In these cases of effusion into the joint it is my practice to use a long posterior splint and bandage with semi-elastic bandages, immobilizing for various periods. Some of them will take as long as two months before the joint can be actively used without recurrence of fluid, and some require only two weeks.

DR. GREER (Closing): As far as the technic of the knee joint is concerned, it does not matter at what point the needle is inserted. As far as splinting, aspirating and immobilization is concerned, in my experience better results are obtained if the joint is immobilized for two weeks. From a comparison of the two methods during the past year I will cite two cases: one was placed on a posterior splint with a large padding of cotton for twenty-four hours, and then active motion used; the other was in a cast for two weeks. The former required aspiration two or three times more than the latter.

Dr. Kennedy's question about rupture of the internal ligament; I think it would be a good thing if every doctor would realize the necessity of going in there immediately, and would do it.

THE TREATMENT OF PNEUMONIA

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(Read before the thirty-eighth Annual Meeting of the Arizona State Medical Association, at Prescott, April 18 to 20, 1929,)

While, from the pathological standpoint, we recognized different forms of pneumonia, such as lobar-pneumonia and broncho-pneumonia, and, from the etiological standpoint appreciate the varieties of the invading organism, no attempt will be made to observe these types in discussing the treatment of the disease, except as it concerns the treatment of the disease caused by Type I pneumococcus.

Absolute rest is the paramount consideration in the treatment of every case of pneumonia, regardless of its exciting cause. From the very moment the physician enters the sickroom of a suspected case of pneumonia, the thought which should be continuously uppermost in his mind is, "How can I give this patient the greatest amount of rest?"

When one considers the nature of this disease, it is quite clear why rest is the keynote of treatment. In pneumonia, as in every other bacterial disease, the final outcome depends on the relationship between the virulence of the infecting organism and the resistance of the tissues of the host. With a single exception, to be noted later, there is no specific treatment of pneumonia. In other words, no substance has yet been discovered which directly affects the organism. Consequently, all our efforts must be directed toward conserving and building up the resisting powers of the host. Many years' experience has proved that rest conserves and builds up the resistance as no other mode of therapy does.

Rest is too often a vague term in the mind of the physician, connoting, in a case of pneumonia, simply "going to bed."

Rest means a comfortable bed, well made and well cared for. It means competent nursing. It means mental quiet: the exclusion from the sickroom of unnecessary visitors, except for such brief moments as will afford comfort to the patient.

Rest means placing the patient in such a position as to give every advantage to efficient respiratory function. This has been found to be the semi-recumbent, or so-called orthopneic, position. For the same reason, it is imperative to remove conditions which interfere with the diaphragmatic and the costal movements, such as abdominal distension, gross pleural effusions and pleural pain. In examining the chest, never allow the patient to sit up; never permit him to turn himself from side to side; always insist that he be turned by the attendant. Frequent examinations are exhausting to the patient and should be avoided.

FRESH AIR

The ideal place for the care of a patient with pneumonia is a large, well ventilated room with an adjoining porch to which the bed can easily be moved without any effort on the part of the patient. He should be kept on the open porch practically all the time, being moved in at frequent intervals for baths, etc. The stimulating effect of cold air on the face obviously increases the depth of the respirations and it has also been found that the B. P. is from 10 to 20 mm. higher in the open air than in a room, even though the room is well ventilated.

If the patient is in the open air, it is especially important that the bed be so prepared that, no matter how cold the day or blustering the wind, the body will not be chilled or inadvertently exposed. An excellent way to accomplish this is to spread over the springs a blanket large enough to extend well beyond the sides and foot of the bed. Over the blanket is spread a rubber sheet or paper. Upon these two materials is placed the mattress, upon which the bed is made in the usual manner, with the quantity of coverings adapted to the weather. Now, over this bed the rubber sheet is drawn up and folded, as one would make an envelope, and secured with pins, thus enclosing the whole bed. If these ideal conditions are impossible, the patient should be in a large, well ventilated room and the air kept actively circulating, if necessary, by means of fans.

CARE OF THE BODY

This important detail is too frequently neglected, especially when a trained nurse is not available. The physician should, personally, instruct the attendant exactly how the

patient's comforts should be cared for. A padded cotton jacket should be worn, closed by tapes tied over the shoulder and under axillae. This keeps the chest warm and can be easily removed for examination of the chest, without disturbing the patient.

A tepid sponge bath, using a mild soap, should be given daily. This may be done between the blankets, exposing one part at a time. The care of the mouth, coated tongue, sordes upon teeth and lips, is of importance. Cotton swabs on applicators, wet with a solution of boric acid (two per cent) should be used on the tongue, cheeks, lips and teeth. Half-strength peroxide of hydrogen is very useful. When the mouth is very dry, equal parts of albolene and a two per cent solution of boric acid with lemon juice added, is effectual. The nose should be kept free of obstructions. Dried secretions may be softened by sweet oil or vaseline.

DIET

During the acute stage, the actual amount of nourishment is of comparatively little importance. The patient needs liquids and, for this reason, an effort should be made to see that he receives at least three quarts in the twenty-four hours. Fruit juices are refreshing and aid the patient in taking the required amount of liquids. Solid food is unnecessary. Soups and gruels, milk, either straight, peptonized, or diluted with lime water, eggs, either soft-boiled or the whites shaken with ice, ice cream, malted milk and custards are suitable. The food should be given at regular intervals, every two or three hours during the day. He should not be disturbed at night to take nourishment. If food is distasteful to a patient, discretion must be used in forcing it. During convalesence, semi-solid and solid food may be permitted very gradually. Food does not cause the distressing abdominal distension which is frequently present. This is probably due to a toxic paralysis of the bowel and cannot be combatted by mere dietary regulation.

BOWELS

The patient should be given a cathartic at the beginning of the treatment. Calomel may be given in ¼ gr. doses every fifteen minutes, for six doses, followed, two hours after last dose, by a saline. The bowel should be emptied every other day by enemata or salines but no drastic purging should be permitted, as it may induce exhaustion.

Meteorism occurs in over half of the cases and often very early in the attack. This not only encourages fermentative and putrefactive changes but also, by pushing up the diaphragm, encroaches upon the respiratory space; encourages atelectasis and embarrasses the heart action. Relief may be afforded by catharsis, by enemata, by turpentine stoupes (1 oz. to 1 qt. of hot water) or by inserting a rectal tube.

FEVER

It is generally accepted that fever is a protective reaction and, unless hyperpyrexia is present, no measures should be taken to control it. For the control of high temperature, hydro-therapeutic agents should be used. Cold sponging every three hours, followed by an alcohol rub, is probably one of the best measures. Antipyretics have a depressant effect on the vital centers and, except in extreme cases, should not be used.

DRUGS AND OTHER MEASURES

The all-important indication is to support the circulation. However, as a result of this conception, pneumonia patients have had poured into them all the drugs which have been thought to have any action on the heart or vessels and, too frequently, they have been administered in a routine manner without any regard to indications.

Digitalis: Digitalis is unquestionably the most dependable drug to support the heart action. It is probable that circulatory failure is due to a direct toxic effect upon the myocardium and damage to the muscle arising from oxygen want. This leads to a decreased cardiac systole, a deficient circulation of blood and vasomotor paralysis. The influence of digitalis under these conditions is probably more prophylactic than curative. It should, therefore, be given early during the course of the disease, before circulatory failure has developed rather than after. Moderate doses throughout the disease are indicated, rather than massive amounts when the circulation is already showing signs of failure. When given in moderate doses, rapid digitalization can more easily be effected should need arise.

Caffeine Sodium Benzoate: For acute circulatory failure, caffein sodium benzoate, gr. 5 hypodermatically, is especially valuable, as it stimulates the vasomotor center, the point apparently most weakened.

Adrenalin: Adrenalin, 10 minims of 1 to 1,000, is often useful in promoting better tone, raising the blood pressure and tiding the patient over an acute failure. However, its action is very transitory.

Strychnine: Strychnine is a drug having little or no value in the treatment of pneumonia and, on account of the increased nervous irritability and excitement which it induces, it is likely to be absolutely harmful.

Morphine: Morphine is, on the other hand, one of the most useful drugs we possess in the treatment of this disease. The importance of rest and quiet has already been emphasized and, in patients who are restless, using their strength up in tossing about and coughing, there is no other drug which will

bring about such repose and conservation of strength and energy. It relieves pain and, in many cases with delirium, produces rest and quiet. It is best given hypodermatically in small doses, gr. 1/8, 1/6, with atropine gr. 1/150, as often as required to meet the conditions present. Codeine may be substituted for morphine in the milder cases.

When sleeplessness alone is present, without nervous excitability, veronal, suphonal, or trional may be used. However, during the acute stages of the disease, if any sedative is required, morphine or codeine will usually be found to be the most efficient.

Glucose: Glucose, it is believed, has a distinctly favorable action on a toxic heart muscle and, in addition, helps to maintain the carbohydrate and glycogen reserves; 250 c.c. may be given intravenously, as twenty per cent glucose solution in normal saline, once or twice daily, very slowly.

Oxygen: The presence of cyanosis calls for prompt treatment. It can be made to disappear or become much less pronounced by the early and proper administration of oxygen. This aids in prolonging life until the immunity mechanism is able to accomplish recovery. The ideal method of employing oxygen is in a specifically constructed oxygen chamber, but this is, of course, restricted to hospitals. Several forms of portable tents have been devised. They consist of a tent made of gas-proof material, with windows in it, which may be attached to the patient's bed. The ventilation of the tent is accomplished by an external, closed circuit which provides an oxygen-rich atmosphere freed from carbon dioxide, heat and excess moisture. The circulation of the air is carried on by a fan driven by a small motor. At the present time, there is no ideal method for the administration of oxygen, by the general practitioner, in the patient's home. The best of the methods ordinarily available to the physician is by the use of a nasal catheter. A long rubber tube leads from the oxygen tank. The catheter is lubricated with cocaine ointment and pushed back through the nostril within half an inch of the posterior wall of the nasopharynx, three inches from the tip of nose is the usual length. Five to ten bubbles per second from four liters per minute, can be easily tolerated. Lacking a meter, give as much as patient can bear.

Much experimentation has been done in an attempt to find some agent that would have the specific property of preventing the growth of pneumococci in the body. Quinine and its derivatives have received considerable attention. The latest is optochin, which has a specific bactericidal action on the pneumococci in the test tube and which, when administered to animals, has caused the disappearance of pneumococci from the blood. However, a careful clinical study carried out by the Rockefeller Institute has been disappointing.

The greatest advance in the treatment of pneumonia has been the development of a specific anti-serum for Type I pneumococcus infection. In cases treated with this serum, the mortality has been lowered from twenty-five to ten per cent. The results obtained are in direct proportion to the speed with which the specific organism has been identified and the specific serum administered. It is best given very slowly into the vein, using 75 to 100 c.c. for the initial dose. The size and interval of the subsequent doses is determined by the condition of the patient. As this procedure requires highly technical laboratory facilities, which are often not immediately available to the general practitioner, the use of serum is not practical at the present time.

CONCLUSIONS

It is desired to stress the following points in the treatment of pneumonia:

1. Absolute rest;
2. Treatment on an open porch;
3. Sufficient fluid intake;
4. Support of the circulation;
5. Early administration of oxygen for cyanosis.

DISCUSSION

DR. S. C. DAVIS, (Tucson): I wish to compliment the doctor on his excellent paper. After such a thorough review of the subject, there is not very much now to mention. One point he stressed was rest. The care that he takes in looking after the little things for the comfort of his patient, makes it very plain that the doctor sees that his patient does have rest. For a number of years, pneumonia carried with it a horror, we realized that we were dealing with a disease that carried a large mortality. In the last two years, and especially during the last year, we have not had the same fear of the disease, whether or not because our cases were more favorable for treatment or we were dealing with a milder type of pneumonia, or whether it was the management of these cases.

The old routine of giving digitalis from the time the diagnosis of pneumonia was made, I believe, is falling a little bit out of use. During the last two years we have not followed that routine. Recently there was an article published which stated that the use of glucose given at the proper time, as mentioned by the doctor in his paper, was much better than the use of digitalis. Since we have discontinued the use of digitalis routinely, we feel sure that our results have been better. One drug that we have now to rely upon for the heart, is adrenalin. Some years ago we began to give adrenalin in drachm doses by mouth. Some will say, at once, that no results could be obtained by mouth. Possibly that is true up to a certain point. We have recently given adrenalin at the proper time; we consider that time is when there is beginning heart failure, signs of cyanosis, or as early before that as we can realize the heart needs help. We feel that the thing that kills in pneumonia is, possibly, toxic heart, caused from the anoxemia that

accompanies pneumonia. Just what adrenalin does besides support the heart, I am unable to explain, but we feel that we have saved a number of lives by the use of adrenalin, given in doses of from ten to fifteen minims, hypodermically, every two or three hours, as needed.

One other remedy was called to my attention during consultation, a few years ago, by Dr. Watson, our president. This is the use of chlorides. He said a doctor in Kansas used chlorides, that is, in all cases of pneumonia, with very good results. Since that time I have given chlorides in all cases and the only thing I use besides salt is the management for rest. We feel salt has served us well. When the peripheral circulation becomes engorged and we have evidence of failing heart, often the withdrawal of 500 c.c. of blood will give a great deal of rest and comfort.

DR. H. K. WILSON, (Holbrook): The doctor just mentioned one thing that interests me because I have followed adrenalin from the time the old extract was used. There is a lot of discussion as to whether it is any use at all given internally. I had hay-fever in Chicago for some time, which was greatly relieved by the use of the extract. I occasionally have a little hay-fever in Arizona and take two doses of adrenalin, with much relief. To prolong the action of adrenalin, if you will add 5 to 7½ minims of pituitrin, you will get better results. You certainly can get results by mouth, but the dose has to be increased two or three times.

With regards to chlorides: ammonium chloride appeals to me as being better than sodium chloride. It is more stimulating and I have used it very largely in cases of pneumonia with fairly good results.

DR. H. D. KETCHERSIDE, (Yuma): I should like to ask a question or two. The doctor spoke of death being due to heart failure, but in a great many cases we have seen it due more to respiratory failure, in that pulmonary edema prevents oxidation, and the problem is how to get the oxygen directly into the blood. Some have given oxygen directly into the veins. I should like to know if the doctor has had any experience in that line, and what he thinks of it.

Another thing I should like to ask about is distention, whether it is justifiable to give hypertonic saline to relieve the distention. It apparently relieves a good deal of toxemia.

DR. ROBERT FRANKLIN, (Glendale): I am particularly interested in digitalis. In an influenza epidemic, I was privileged to observe two wards under two commands. In one, early digitalization in pneumonia was used, and not in the other. I was convinced that early digitalization was not as successful as formerly had been thought. This, I believe, was due to overstimulation of a right ventricle against a consolidated lung. You are whipping a horse before he gets to the hill; you are tiring a right ventricle before it gets to the point of cardiac failure, and at the time you need this stimulation you have a tired heart.

DR. W. C. TODT, (Kingman): Nine out of ten get well, and the tenth one the devil himself can't save. Thirty years ago I used carbonated water and I still use it. I get better results from it than anything else. The death rate seems to be as great now as fifty years ago.

DR. W. W. WILKINSON, (Phoenix): Any man who has practised medicine has had plenty of experience in lobar pneumonia. I want to compliment Dr. Allen on his paper, and to speak a word as to preventive medicine. We don't have much pneumonia, 'flu', pleurisy, colds and rheumatism during the summer. We are tremendously busy with this during the winter. We drink quantities of water during the summer and perspire freely (especially in the south), have a comparatively light diet, plenty of sunshine and air. I think the use in winter of the artificial sun bath with liberal use of water—three to four quarts daily for the average individual—would maintain the resistance established during the summer. Personally I use the mercury vapor lamp.

DR. JOHN W. FLINN, (Prescott): Speaking for the program committee, we quite agree with Dr. Todt that statistics show the death rate now is as great as fifty or sixty years ago. That is the reason we asked for this paper. We feel that there is something radically wrong when no better results are now obtained from the treatment of pneumonia. It seems to me the strong part of the paper is that it sets forth a definite technic for the treatment. I take it that when the surgeon gets poor results he says there is something wrong with his technic and immediately sets about to remedy it. That is what Dr. Allen has done in this paper.

With regard to digitalis, I think Dr. Franklin got the wrong idea of Dr. Allen's treatment. I believe he suggested that digitalis be given early in small doses, with the idea that when the crisis approaches it is easier to deal with.

DR. FRANK J. MILLOY, (Phoenix): Two things in pneumonia are the failing heart and increasing pulmonary congestion. Dr. Allen has said that the usual position is the recumbent. When you stop to consider, the recumbent position is most likely to develop increased hypostatic congestion and is also the most embarrassing position for the heart. Some patients, not all, may be turned face down. It is surprising how many can be turned into this position. If this is done, hypostatic congestion seldom develops and the heart works more easily. It also relieves gastro-intestinal symptoms.

DR. JOHN ALEXANDER, (Ann Arbor): May I suggest the use of ephedrin sulphate instead of adrenalin, in impending circulatory failure, in that the action seems to be prolonged. In postoperative pneumonia, there has been definite advance in the last year. So many patients die, although many get well. It has been found in Cornell Medical School that the true postoperative pneumonia is due to obstruction of large or small bronchi, or both, by tenacious mucus plugs. If these are caughed up or aspirated, the patients get well. There is, constantly, postoperative pneumonia, which occurs in a few hours after operation or in a day or two. Sometimes the patient will come back from operation that has been a simple one, will apparently be doing well, and suddenly develop a high fever and cyanosis. If given inhalations of oxygen or steam inhalations and urged to take deep breaths, the patient will cough up from one to three ounces of mucus in a few minutes and breath sounds, which will have been absent, will return. Repeatedly in the past year I have seen patients to whom this has been done and the crisis appears in twelve hours from the time this treatment is instituted. The cure will be almost constant.

DR. DONALD JACKSON FRICK, (Los Angeles) The question of digitalis in pneumonia has interested me a great deal. During the war, we had a big service in Louisiana. Before I took charge of the medical service, the chief of the service was very keen about digitalis and did not give it in any small doses in the beginning. He gave as much as 5 or 6 c.c. intravenously, and kept it up for the first twenty-four to thirty-six hours. When I went down there, we discussed this question and I said I thought they were fairly large doses. He stated that was perhaps true, but he did not believe that digitalis was very good digitalis and he wanted something to take effect.

You can poison people with digitalis but I do not believe it has much effect otherwise, unless in

tremendous doses. I think the value of digitalis in pneumonia is entirely in older people who have some degree of damage to the heart muscle, in which we wish to slow the heart to increase the blood supply through the coronary arteries, thus making more efficient hearts. I think, in young healthy people, digitalis has not much effect unless they are poisoned by it. In older people, as the disease progresses, they may fibrillate or get definite changes of that kind, and you should be in a position to have enough digitalis in their systems so that a little more can fully digitalize them.

Unfortunately, our mortality has not fallen much in the last years, with all our experience. In Type I pneumonia, we have made an advance and are hoping for a serum for Type II and Type III which may be definitely effective. The percentage of mortality has been around 30 per cent in virulent epidemics and 15 to 18 per cent in the mild type of epidemic.

DR. JAMES H. ALLEN, (closing): I wish to thank the doctors who discussed this paper. It has been very instructive to me. I was particularly interested in the discussion of the value of digitalis. I use small doses during the course of the disease from the beginning, and then, if the need arises, it is easier to get rapid digitalization.

Regarding pulmonary edema, I believe, in a number of cases, it is due to acute circulatory failure. The administration of oxygen will frequently tide them over before the edema becomes well developed. After it has become well developed, it is rather hopeless to do anything.

About the recumbent position. What I said was "semi-recumbent position", and I believe, if you go through a hospital where there are a number of heart cases, you will find practically all cases with any dyspnea in the so-called "semi-recumbent", or othopneic, position. That is the position they naturally take.

THE EYE IN SYSTEMIC CONDITIONS

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(Read before the thirty-eighth Annual Meeting of the Arizona State Medical Association, at Prescott, April 18 to 20, 1929.)

The eye is not an organ to be set out in the garden of Science alone, to be cultivated and treated as a separate, distinct entity. It is not only the index of the soul but it is the index to the entire body. If the index is in a language unknown to us, we cannot read it, but if we can read it easily, we can soon turn to the pages that give us the full text of the subject under consideration.

Go with me to watch the ophthalmologist examine your patient. Let us say the first patient has a very red, inflamed eye. If this is conjunctivitis, move the conjunctiva and the blood-vessels will move with it; if it is glaucoma the tension will be high, that is, the globe will be harder than normal, and the pupil will be dilated; if it is scleritis or iritis, the blood-vessels will not move with the conjunctiva when it is moved. They will be deeper in the tissue and will "dip" into the sclera four or five millimeters from the cornea. In scleritis the color is a bluish red. Scleritis is a symptom of, or is caused by, rheumatism, gout, syphilis or tuberculosis. In iritis, the cornea is hazy, the pupil contracted and will not react to light or accommodation; in glaucoma the pupil is dilated and does not react readily to light or accommodation. Let me digress here to say that the treatment for glaucoma will do much harm to an iritis and the treatment for iritis will do much harm to glaucoma, thus the price of making a mistake. In iritis one can often see blood-vessels, and sometimes blood, in the iris. In glaucoma, the anterior chamber is very shallow and the patient has halos or rainbow colors around lights. With both diseases the patient suffers intense pain.

IRITIS

Iritis is always a symptom of some grave constitutional disease.

Your opthalmologist will tell you that if you have an iritis with overfilled vessel loops, which are bright red and located at the margin of the iris, it is probably caused by acquired syphilis. If there is one, or more, yellowish, reddish-yellow or reddish-brown nodules the size of a hemp seed to a small pea, it is almost pathognomonic of acquired syphilis. It usually appears from the second to the seventh month of the disease. My associate, Dr. Robb, has a patient sent to him a few months ago with this condition. He promptly sent him back to his physician with a diagnosis of syphilis. His physician later found a four plus Wassermann. We occasionally see a gumma in the iris which comes on in the tertiary stage of syphilis. Iritis in children from the first to fifteenth month, is usually caused by hereditary syphilis. The cause of more than forty per cent of all cases of iritis, is syphilis.

In iritis in children and young adults, ages five to twenty-five, there may appear a grayish-red or a yellowish nodule with many very small nodules around it or, in some cases, a single granuloma. This might be mistaken for syphilis, but the nodules do not appear or disappear so quickly as in syphilis, that is, it is more chronic. Here the tuberculin or von Pirquet test will help to solve the problem. DeSchweinitz believes we have primary tuberculosis of the eye, but Kuckman, of Berlin, has shown that tuberculosis of the eye, whether iritis or keratitis, comes mostly from tuberculosis of the glands and especially from tuberculosis of pretrachial glands. Franceschelti, of Basel, has found that it also comes from tuberculosis of bones.

We have iritis from gonorrhea, either from gonorrheal conjunctivitis or by metastasis from the urethra. The latter may come on after the urethra has been cured.

Iritis following a corneal ulcer, as in smallpox or diphtheria, is apparent.

We may have iritis in gout, and acute rheumatism, but not in muscular rheumatism.

PUPIL

Anisoceria, that is, one pupil large or the other very small, is seen in progressive paralysis. If very small and not round look for tuberculosis or general paresis. This uneven pupil may be taken for an eye after iritis with posterior synechia. In general, the pupil contracts in a bright light, when looking at near objects and in old age. Immobility of the pupil is of most importance in tuberculosis, cerebral syphilis and in general paresis.

Blue sclera tells us that the patient has brittle bones. This was ably discussed in a paper by Dr. George Shields, at a meeting of this society, in 1927.

CORNEAL DISEASES

Phlyctenular conjunctivitis is characterized by one or more grayish or white elevations on the cornea, on or near the limbus. A fan-shaped leash of blood-vessels extends from it onto the conjunctiva. The white spot often breaks down to form an ulcer. These occur in children of low vitality, either under-nourished, scrofulous, or with tuberculous tendencies. Only a few weeks ago, a woman brought in a beautiful well-nourished girl, five years old, with one of these phlyctenules. As usual, the mother asked, "what is the cause of this condition?" I answered her by asking if she and her husband were both well, or if any tuberculosis or constitutional disease existed in uncles or aunts or other members of the family. Then she told me that her husband was in bed with tuberculosis.

When trauma and ulcers can be eliminated, about eighty per cent of phlyctenular keratitis is caused by inherited syphilis. About ten per cent is caused by tuberculosis. In these keratitis cases deafness is often found. In taking the histories, it has been found that about fifty per cent of syphilitic children die. Keratitis comes on in children of ages from five to fifteen.

DIABETES

Look for diabetes when your patient comes in with a sudden change of refraction, as a marked myopia when the same patient had normal vision or hyperopia before. If there is a rapid decrease in blood sugar, as after energetic treatment, the refraction changes to hyperopia. These changes may be as much as seven or eight diopters and are probably due to osmotic pressure involving the lens caused by concentration of the blood and tissue fluids with sugar content. These refractive errors return to normal, or near normal, when the carbohydrate metabolism has been re-established.

Ambliopia is another symptom or complication of diabetes. This ambliopia is clinically identical with nicotine or alcohol ambliopia. Here let me say that diabetics cannot tolerate tobacco as well as non-diabetics, therefore, a diabetic may develop a nicotine ambliopia when the same amount of nicotine would not affect a non-diabetic. Failure in vision is accompanied with central scotoma for red and green. There is a rapid improvement on the disappearance of acetone and oxybuturic acid from the urine.

If an iritis comes on in a diabetic it is usually bilateral and is characterized by great swelling of the iris with gelatenous exudate and is amenable to carbohydrate-free diet.

Ocular palsies are fairly common in diabetics. Diplopia is caused mostly by involvement of the sixth and third nerves. These may occur as a peripheral neuritis or from a hemorrhage, either centrally or any place along the course of the nerve.

The opthalmologist now makes a fundus examination and he will find the following:

(1) Rhetinitis in ten per cent of the hospital cases of diabetics, and it is believed that the percentage is higher in those not in the hospital. Here he finds white spots, hemorrhages, papillitis and other signs of inflammation. The white spots are not arranged in stellate form around the macula, but are arranged mostly on the central region of the retina and farther away from the macula than in Bright's disease. Sometimes they are on the other side of the disc from the macula. The patches have a rather sharp edge and a soapy or waxy appearance. The hemorrhages are rather petechial in character. Abundant hemorrhage is comparatively rare. The prognosis is better in diabetic retinitis than in albuminuric or arteriosclerotic retinitis.

(2) He sometimes finds lipemia retinalis. This is a condition in which the fat content of the blood is at least four per cent and the retinal vessels have characteristic salmon or milky appearance. The arteries are hardly distinguishable from the veins. They lose their cylindrical contour and have the appearance of being overfilled and become white near their peripheral. When this condition occurs, the prognosis is very grave.

The best understood of the ocular complications in diabetes is cataract. It is found in four per cent of all cases coming to operation. These statistics were gathered before blood-sugar tests were routinely made, so the true percentage is much higher than this. True diabetic cataract is seen ni young subjects and is bilateral, beginning in the posterior layers of the lens. The juvenile

type is characterized by great rapidity of development, often a few weeks. The cataract appears blue-white, milky and homogeneous. The characteristic appearance of the adult type during the early stage is the diffusion of the opacities between posterior primary embryonic neuclear and the cortical demarcation, instead of the usual senile spike-like and cleavage lines. Before operating on a diabetic patient, it is better to have him rest, and on a diabetic diet, with an effort made to eliminate at least a part of the hyperglycemia and antonemia. Benedict says not to give Insulin after a cataract operation for at least four weeks, because it produces hemorrhage in many cases and thereby causes the loss of an eye and spoils the effect of an otherwise skilful operation.

Albuminuric retinitis is more characteristic. If we find the white patches arranged in the form of a star around the macula it is almost a pathognomonic sign of albuminuric retinitis. In this same eye you will have other white patches, quite small in some eyes and quite large in others, even to "snow bank" in appearance. These white patches may be distributed over the retina generally, but are usually found between and above or below the macula and the papilla. Along with the white patches may be seen hemorrhages which usually occupy larger areas than in diabetes. It is true that we cannot always tell whether a white spot is an albuminuric or a diabetic retinitis, but this we do know, that they are warnings to show us that danger threatens. Our duty, therefore, is imperative, just as it is in a hemorrhage in the retina. Make a thorough examination of the body and especially of the blood and urine. Here, too, neuroretinitis may be observed. The nerve head is swollen, very red, covered with fine blood vessels and has a halo around it. The arteries are smaller than normal, sometimes almost obliterated. The veins are very broad and tortuous. This may be taken for a chocked disc. The diabetic neuroretinitis is different from the albuminuric retinitis in that it does not have the stellate white spots, and, as mentioned above, the spots are more diffused over the retina.

How does all this help? It help in prognosis. In albuminuric retinitis and neuroretinitis, except in albuminuria of pregnancy and scarlet fever, make your prognosis grave. The life of such patient is usually eighteen months, or less, rarely more than two or three years. The prognosis of diabetes is better now since Banning's discovery.

HEMORRHAGE

When injury, high myopia and glaucoma can be eliminated, hemorrhage into the eye is always a sign of a general disease. It is a warning that something is out of order in the general organism. Fine striate hemorrhages which radiate from the papilla are superficial, while round or lumpy ones are in the deeper layers of the retina. Little shuttle-like hemorrhages with a white spot in the center are usually caused by diseases of the blood such as anemia or leukocythemia.

Hemorrhages in nephritis are found mostly in granular atrophy of the kidney. Hemorrhages are found in infectious diseases, such as influenza, typhoid, malaria, miliary tuberculosis and in syphilis. The most important is hemorrhage in arteriosclerosis, because of its prognostic value. You will recognize arteriosclerosis in the eye by fine corkscrew arteries, a compressed vein where an artery crosses it and a dilatation of the vein on the distal side of the artery. Arteriosclerosis here is an early sign of the same condition now existing or soon to come on in the kidney and brain. A hemorrhage into the eye in this disease, in fifty per cent of cases, is a forerunner of a hemorrhage into the brain.

I will not discuss, further, hemorrhages into the eye, because this subject was so ably discussed before this Association in 1925 by Dr. D. F. Harbridge.

In optic atrophy look for syphilis, tuberculosis or paresis.

The last, but not the least, to be discussed, is syphilis. Really, I will not discuss this subject but only mention a few of the most important things seen in the eye in this disease. One could write a whole paper on this subject and then not exhaust it. So I will say, if we have an iritis with very red granules at the margin of the pupil, look for acquired syphilis in the early months, usually about the second to the fifth months. If we find phlyctenular kerititis, look for inherited syphilis. This usually comes on in children. In some cases of papillitis and retinitis look for syphilis. Sometimes hemorrhage into the eye is caused by syphilis; so is optic atrophy, retinitis pigmentosa. The salt-and-pepper fundus is almost pathognomoinic of inherited syphilis. Many other diseases in the eye are caused by syphilis. So, when in doubt, look for syphilis.

There are many other conditions that could be discussed in this paper but time and space forbid. Let us hope that others may "carry on."

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DISCUSSION

DR. F. C. JORDAN (Phoenix): What is the prognosis in hereditary syphilitic keratitis in children?

DR. C. R. K. SWETNAM (Prescott): I don't know anything about the eye. Dr. Bailey has given us something that we can not get from ordinary textbooks, the tabulation of some of the conditions that we find, especially those of us who are not doing eye work. I think, from the standpoint of the general practitioner, it is a case of knowing just enough about it to know when to send the patient to a specialist. I should like to hear the men who are doing work in diabetes and nephritis give the relationship between their work and Dr. Bailey's.

DR. C. N. PLOUSSARD (Phoenix): I should like to ask Dr. Bailey to mention again the causes of unequal pupils in children.

DR. BAILEY (closing): Answering Dr. Ploussard's question, the immobility of the pupil is most important in tuberculosis, general syphilis and paresis.

The prognosis in keratitis, syphilitic keratitis, is, generally speaking, very good. In children, this usually clears up under some form of iodine, especially hydriodic acid.

Dr. Sharp whispered to me about nephritis in obstetrics. For a long time I have thought that every pregnant woman should have a fundus examination. Sometimes you can look into the eye and see nephritis coming before it shows up very much in the urine. Any man is supposed to be able to look into the retina and see white spots. After a long time these turn dark and are covered with a more or less pinkish color. In the nephritis of pregnancy, after the baby is born and the woman receives proper treatment, the nephritis usually clears up. This is true a great many times in scarlet fever. Exclusive of these, they usually do badly.

One of a number of cases sent me by general practitioners, had stellate degenerative processes in the eye. I told her doctor that this woman would probably not live very long, and it is true that she did live less than eighteen months. It has been my experience that they do not live much over eighteen months after these white spots develop in the retina.

Pregnant women do not have such large white spots and they usually clear up with proper treatment after the baby is born. I do think every pregnant woman should have a fundus examination.

THE COST OF FREE ADVICE. TUBERCULOSIS IN AN INDUSTRIAL GROUP

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(Editorial note:—The following article appeared in the April, 1929, issue of the Journal of Industrial Hygiene, page 107. It is of such pertinent value to industrial organizations and medical men working for such organizations in the southwest, that the entire article is reprinted herewith.)

One of the greatest obstacles to the effective control of tuberculosis is found in the late diagnosis of the disease. The reason for delay in diagnosis may be the ignorance or the indifference of the patient, the cost of medical service, or the diagnostic failure of the physician. This paper presents briefly the results of the study of a small group of tuberculous patients whose cases, in spite of conditions which would appear to favor prompt diagnosis and favorable outcome, nevertheless were diagnoses late.

The group was selected for study from a much larger number of sanatorium patients because our information concerning their mode of life, the medical facilities available to them, the cost of their treatment, and their condition after discharge is unusually complete. These patients were the tuberculous employees of one division of a railroad and were treated during the past four years. We received them without selection shortly after their tuberculosis was diagnosed. They constituted for the period of time under consideration the tuberculosis problem of a rather stable industrial community of some 15,000 workers—a community none the less though it happens to be stretched along some hundreds of miles of right of way. They were all white, and either skilled workmen or office workers. The company employs for common labor mostly Mexicans, with some negroes and Japanese, and the twenty-eight of these who fell ill of tuberculosis during the four-year period under consideration are not included in this analysis. They are, however, included in estimating the annual tuberculosis admission rate, which is approximately one case to every 800 persons employed.

These patients lived and worked in a good climate. Being railroad men, they earned an adequate living wage, and could afford decent quarters and ample food. They were all members of a hospital association, to which they contributed a small sum from their monthly pay; they were entitled to consult the company doctors without charge, they received certain medicines free, and, if tuberculous, their sanatorium bills were paid for a period varying with the length of their employment. The railroad company takes an active interest in this hospital association and helps its operation in many ways. Sick men get free transportation, their jobs are held open for a reasonable time or easy work is found for them, if necessary, when they recover. There is a well-equipped central hospital where diagnostic laboratory work is done, also without charge. If the patient is willing to accept institutional treatment, his diagnosis is followed by transfer to the sanatorium within a short time unless his condition forbids the jour-

ney. The expense of medical service, therefore, did not delay diagnosis and treatment in any case of this series.

This corporation has a good medical staff. Its local surgeons, selected of course for their surgical ability, are among the best in their respective communities. It carries on a safety first campaign for the prevention of accidents, and is considered to be, in general, efficient.

During the four years covered by this analysis, thirty-eight tuberculous employees were discharged from treatment. Their classification when received at the sanatorium, according to the National Tuberculosis Association standards, was:

	Number	Per Cent.
Far advanced	33	87
Moderately advanced	3	8
Minimal	2	5

There are now in the sanatorium under treatment six patients, and five of these are far advanced—practically the same proportion. Of this whole group of forty-four patients, then, thirty-eight, or more than 86 per cent, were far advanced in their disease when admitted; fifteen were classed as far-advanced "C," with "severe symptoms: marked impairment of function, local or constitutional;" which implies an unfavorable prognosis.

The duration of treatment of these far-advanced patients varied from less than 1 month (when death occurred) to 27 months, and it averaged 10.3 months. Of the thirty-three far-advanced patients, three, or 9 per cent., died in the sanatorium; ten or 30 per cent., were discharged unimproved; twelve or 36 per cent., were improved, and the remaining eight, or about 24 per cent., were quiescent, apparently arrested or arrested.

The follow-up of these thirty surviving far-advanced patients, from one to three years after their discharge, is interesting. Two (one was unimproved when discharged) have not been traced. Eleven, or about 37 per cent. of the number, have died, eight of these were unimproved when they left the sanatorium. Six are living but are still under treatment. The remaining eleven report themselves able to work, but only seven of these are working for the company that formerly employed them. Excluding one suicide, we know that thirteen out of thirty-three far-advanced patients have died of tuberculosis within four years after their admission—a mortality of nearly 40 per cent., as compared to eleven, or 33 per cent., who are able to work, and but seven of these are back at their former jobs. It cost the hospital association about $32,300 to treat the far-advanced cases; or $4,600 for each trained employee returned to his work.

In striking contrast are the results obtained in the treatment of the three moderately advanced patients in this series. After a period of hospitalization averaging 11.3 months they were discharged as arrestetd or apparently arrested; two are now working for the company, the third is about to resume work. It cost about $1,050 each to repair these men. Similarly, excellent results were obtained with the two early cases. They were returned to work after a few months of treatment, apparently arrested, at an average cost of $450 each.

Clearly, an early diagnosis was of great advantage to employer and employees alike. These people were of good average intelligence, they had an organization designed to protect their health, and its services were to be had for the asking. Why then was the recognition of pulmonary tuberculosis so long delayed? A study of the histories of the far-advanced cases returns at least a partial answer. One necessarily makes allowance for some inaccuracy on the part of the patient in relating the onset of his illness; nevertheless, the information in the mass is probably as accurate as any other testimony. In obtaining these histories care was taken to avoid leading questons. When possible, the narrative has been checked up from sources other than the patient, and as a rule it has been found to be substantially correct.

Three patients admitted that they came to the Southwest to obtain the benefit of the climate, and entered the company's employ knowing that they had tuberculosis. They continued to conceal their disease as long as they could. Two of these three are dead; one is working.

Ten patients with chronic tuberculosis of the fibrocavernous type appear to have paid little attention to their symptoms, which were annoying but not disabling. They neglected to place themselves under continued medical observation, though most of them had gone to the doctor from time to time and asked for some medicine for a "bad cold," or for "stomach trouble," these being their words for what ailed them.

Four patients had an abrupt and stormy onset, preceded by few symptoms, or none that they noticed. In these cases the disease must have progressed almost silently to the far-advanced stage, when it suddenly produced disability. Hemorrhage was the first frank manifestation in three of the four. They were, of course, diagnosed at once.

So far as these seventeen patients are

concerned, there was no diagnostic failure. It would have been better for them if they had been required to pass a physical examination before employment; a periodic health examination would have been even more useful. A warning against tuberculosis, with some mention of the early symptoms, might well have been included in the safety first articles which were brought to their attention. In nearly half the cases, therefore, the immediate responsibility for delayed diagnosis rests on the patients themselves and the best preventive measure would have been elementary education in the care of their health.

Seven patients, who had previously been treated for tuberculosis by physicians in the company's employ, relapsed after working for a period of time which varied from six months to as many years, and they were far advanced at the time of their relapse. It does not appear that these workers took the trouble to report regularly for chest examination, nor was any systematic effort made to keep them under observation. It would have been profitable to all concerned if they had been called in for an overhauling, say every three months for the first year and yearly thereafter.

Three men, under treatment for unrelated surgical conditions, developed tuberculosis of the lungs while under medical observation and it was not detected early.

Eleven men had been ailing, off and on, for months or years. If their statements are true, they had repeatedly gone to their local surgeons complaining of frequent colds, chronic coughs, persistent indigestion, loss of weight, lack of endurance—symptoms which should cause a medical man to remember the existence in the world of a disease called tuberculosis. But their sputum was not examined, their chests were not x-rayed; they were given medicine to take; and they were far advanced when the diagnosis was finally made. It may be merely a coincidence that several of this group had been under the care of one surgeon.

In fairness, it should be said that many railroad men, if not completely disabled, will not take time off to consult their doctor at regular hours; they hurry in at the end of the day, or the end of the run, asking for medicine for this or that symptom, take the prescription, and disappear. And it is pertinent to remark that, while a few of the company surgeons are on a salary, most of them receive for their work no tangible compensation other than an annual pass.

It is apparent that free medical service, without special attention to the prevention and detection of tuberculosis, has not availed to control the menace of tuberculosis to this particular industrial group. The purpose of this study is not to fix the blame for this failure, but to find the reasons for it, and, if possible, the remedy. It is hoped that the work, though limited in its scope, will be of some use to others interested in industrial medicine.

The underlying cause of the delay in diagnosing tuberculosis seems to have been the human tendency to overlook the obvious, to ignore commonplace knowledge, and to look for the new. The employees must have encountered consumptives frequently; the train crews of necessity saw them, both alive and dead. The company doctors knew that tuberculosis is still a prevalent disease, fifth among the causes of death and ranking higher than that as a cause of disability and poverty. That they were not shirking work is shown by the operations they did for the elimination of foci of infection, to which they not infrequently attributed the symptoms caused by early tuberculosis. While the total damage from tuberculosis was considerable, the incidence of the disease in the separate groups was not heavy enough to hold the attention of either physicians or laymen.

Admittedly, not all cases of tuberculosis can be detected early, unless recourse is had to entrance examinations and periodic health examinations conducted by experts. But it should be possible to bring about a much earlier diagnosis of most cases, without changing or overtaxing the existing organization. People nowadays are accustomed to have their thinking done for them; they are ruled by advertising. The thing to do in this instance is to advertise tuberculosis. It might be difficult to educate the rank and file of the employees to be alert for the well-known onset symptoms of tuberculosis, but it would be easy to instruct their leaders, the bosses and foremen and superintendents, as one of the ordinary safety first procedues, to send in for examination all workers, whether in field or office, who suffer from persistent cough, failure of efficiency, or obvious loss of weight. The situation should be brought frankly to the attention of the company doctors. While it is not to be expected that a busy surgeon should infallibly search for and detect the physical signs of early tuberculosis, he may reasonably be required to have the sputum of every suspected employee examined, not once but repeatedly if cough persists. Roentgenograms of the chest, made and interpreted by x-ray workers experienced in chest work, should be used as a matter of routine. The cost of the treatment of one hopeless case would pay for many such films. And, in order to insure the co-operation of the

patients, the trip to the hospital for diagnostic work had better be made on the company's time. Finally, the recovered patient when returned to work should be brought in for periodic examinations until the danger of relapse, greatest in the first year and decreasing thereafter, becomes remote. A man worth repairing is certainly worth keeping in repair.

So much for the white employees. The situation in respect of the laborers, particularly the Mexicans, is less hopeful. Our histories of these are as rule not satisfactory; many are illiterate, and we cannot trace them after discharge. Often they do not cooperate well in their treatment; they have a racial lack of immunity to tuberculosis; they insist on living as they did in their own country. Nearly all are far advanced when diagnosed, and recovery is the execption. Eleven of the twenty-eight died in the institution where they were treated. There appears to be nothing to do with them under the present system except to give humanitarian care to the ones that fall sick, and to trust to their enlightenment through the education of their children. If more is to be accomplished, routine physical examination will have to be made a condition of their employment.

But the whites, who outnumber the foreigners, are educable; and the simple measures outlined above, if put into effect among them or in any other industrial group, should be well worth while. For tuberculosis is not prone to attack the sluggard and the shirk; it selects its victims from the energetic workers, those who keep going to the limit of their strength. And such persons, if put under treatment early, are likely to be returned without serious damage.

SUMMARY

This article is a review of the cases of tuberculosis occuring in a group of railroad employees during four years. The annual tuberculosis admission rate in this group was about one case to every 800 employees.

These employees lived in a good climate; they earned adequate wages; they were members of a hospital association and as such were entitled to free medical service, and to free sanatorium treatment.

The detailed study is limited to the white employees, mostly skilled workmen or office workers of whom full histories and nearly complete followup records are available: There were forty-four such patients, of whom thirty-eight were classified as far advanced when admitted to the sanatorium, fifteen of these being rated as far-advanced "C."

The mortality from tuberculosis among the far advanced, within four years of their admission, was nearly 40 per cent. Only seven of the far advanced have gone back to their former jobs.

All the moderately advanced and minimal patients recovered, and are able to work.

It cost the hospital association on the average $4,600 to put a far-advanced patient in condition to resume his former job; the cost for the moderately advanced was about $1,050; and for the minimal, about $450.

Inquiry concerning the reason for delay in diagnosis shows that three patients deliberately concealed their symptoms; ten neglected to consult the company doctor until late in their disease; and four, whose disease had progressed silently, were far advanced at the time of the abrupt onset of symptoms.

Seven were old cases, formerly treated. They had not been under regular observation since their return to work.

Eleven, who had been more or less under medical observation for symptoms suggesting the existence of tuberculosis, were not diagnosed until late. Tuberculosis appeared as a complication in three surgical cases, and gained considerable headway before it was detected.

Free medical service, without special attention to the prevention and detection of tuberculosis, did not control consumption in this industrial group.

The underlying cause of the delay in diagnosis appears to have been the tendency, on the part of laymen and doctors alike, to ignore the commonplace knowledge of the prevalence of tuberculosis, and of its earlier symptoms. They were not "tuberculosis-minded."

It is desirable to bring to the attention of both laymen and doctors, with sufficient frequency and emphasis to make the impression persist, the prevalence of tuberculosis among industrial workers, and the early symptoms of that disease. In a world full of advertising it is necessary to advertise tuberculosis also.

Repeated examinations of the sputum, and roentgenograms of the chest made by x-ray workers skilled in chest work, should become the routine and not the exceptional method in dealing with suspects. Reliance should not be placed on a casual physical examination. Tuberculous patients returned to work should be kept under observation until the danger of relapse becomes remote.

In groups covered by any sort of health insurance, and under even limited control, these measures are practicable and they will more than repay their cost.

DESIRE OR COVETOUS NEUROSIS

PHILIP KING BROWN, M. D.
Medical Director, Southern Pacific Hospital, San Francisco, California.

(Presented at the Pacific Association of Railroad Surgeons, San Francisco, Aug. 24, 1928.)

"Thou shalt not covet thy neighbor's house. Thou shalt not covet thy neighbor's wife nor his man-servant nor his maid-servant nor his ox nor his ass nor anything that is thy neighbor's."

A child of four, in a preventorium, made so great a disturbance over being examined that the ordeal was postponed and a study made of her reactions to her surroundings, without centering any apparent attention upon her. She promptly felt the neglect and, within a few days, she began a series of complaints of pain here and there, so as to have the fullest attention of the doctor, who made of his visit to the other children a game in which the spoiled one had no part. It was interesting to see how she acted each new role of headache, pleurisy, stomachache and bad cold, but, when the doctor's visit was over, so also was the pain.

A daughter of ten became the sister of a child who turned out to be feeble-minded. As the older child passed through adolescence and approached the time when her parents should both have been thinking and planning for her future, her final steps in education and her social life, they were so wrapped up in the handicapped child that they gave these things no attention. The girl grew introspective, sensitive, lazy, constipated, nervous, developed severe abdominal attacks which made her an invalid. She finally grew so ill that she got her share of family attention. Her father was a surgeon and for years his one idea was that surgery might help the girl some way.

A young man has pain two hours after eating and a diagnosis of duodenal ulcer is made. Under adequate treatment he recovers. Later, he writes to the hospital for data in an attempt to connect this disability of 1927 with his service record in France in 1918. He had food poisoning there, and a weak digestion, with constipation, ever since. From three or four sources, through the Red Cross, Veterans' Bureau and various benefit societies, we are approached to assist the case by stretching our evidence to help this man secure a permanent disability rating.

A locomotive engineer of 56 is found to have moderate myxedema and not nephritis, which had been diagnosed elsewhere, causing him great alarm. In a few weeks this dull monstrosity, with his thick lips, coarse, yellow, dry skin, stupid look, almost hairless scalp, suffering with cold, unable to work for months, was converted into a fine-looking, red-cheeked, active man. He was granted a pension for long and active service, but for a year he has hounded the hospital authorities to grant him a permanent disability rating, which is given only when infirmity prevents the man from doing any form of work whatever, and carries with it futher substantial monthly payments. He bases his claim on symptoms associated with nervous instability.

A thousand accident cases come and go in a great hospital like the Southern Pacific's. They vary in seriousness from those which cause death from shock, down the line of fractures of the skull, sometimes depressed, fractures of all parts of the spine, crushed wounds, burns, bruises, sprains, to some that are so trivial that many people with them would give them no consideration. We never take any injury other than seriously. One man may make light of a very grave matter and another make a huge fuss over little or nothing. It is the former case that sets the standard of investigation and care, for he is more apt, in the long run, to be overlooked. One is quite sure that the exaggerated reactor will get all the attention he needs, for we know, often long before the patient does himself, that he is potentially a trouble maker and a damage claim is in the offing, and we must thoroughly study the case to protect ourselves from any form of claim that may arise later. It is not very different from the case of an accident to an automobile and many a camouflaged Ford has been pictured a glorified Rolls Royce when it has been damaged.

It is more or less human to want something for nothing and to covet some extra possession from the source of plenty. People react very differently to the temptation to lean on strong support. The emotionally unstable, the world over, are the makers of difficulty and their inability to fit into human society normally or to adjust themselves to sudden new conditions, even favorable and easier ones, makes them the hard problem in our medical work. They want what they have not, and they never want work. In the case of an automobile accident if an expensive limousine is responsible, or can be made to seem so, for an injury to an old rattle box of a Ford truck on its last legs, it is always a marvelously well preserved and efficient Ford, doing uninterrupted service and only a new truck will be adequate compensation and cure the covetous desire. Should such an antiquated Ford, however, be responsible for an injury to an expensive limousine, the desire never arises in the mind of the limousine owner. He knows the utter impossibility of collecting ten cents damages, so he suppresses his wrath and wastes no

time coveting what he knows he cannot get—even just damages. Our medical problem in all these cases is to prevent the onset of the covetous desire, or to deal with it, if it has already arisen, as promptly and finally as possible. Once it is well established, there is no cure but the final decision of the court, or a settlement which compensates the individual to his satisfaction. If we now go back and recall the situation in the cases used as illustrations, we can see the instinctive and instructive beginning of this sort of neurosis, and how easily it may be prevented or cured.

The child who makes an undue fuss is completely ignored, but learns how others get attention and simulates these conditions. Of course it is play, but it is the mechanism by which thousands of cases go on to serious conditions. The child may make a fuss to get what it wants, the woman may have a brain-storm for the same reason.

The young woman with the strong desire for parental attention developed, quite unconsciously, a mechanism for obtaining it. The cost was great, but it worked.

The war left us thousands upon thousands of grafters on public bounty, drawing compensation for conditions on claims absolutely unwarranted. While we cannot do enough for the youth of the land who really were handicapped by service illness or injury, the shameless imposition of many on the generous government is one of the blots of the war.

Finally, our engineer, rescued from a life of stupor and discomfort, adequately pensioned, able to earn his living at a less demanding occupation than an engineer's life, instead of being grateful, is disgruntled and unhappy and has spent energy enough, trying to get a few dollars more, to have run a small business.

It has been the experience of the Medical Department of the Southern Pacific Hospital that, the sooner an injured man is settled with and put back to work, the simpler the situation. In an experience covering twenty-four years, during which I have examined and reported on a good many cases of traumatic neurosis, I have been called upon to testify in court in only three or four cases, because of this policy of the Company in dealing with these cases if there is any just ground on which the injured man presents his claim. Of the surgical injuries, I cannot speak, but the annual reports of the legal department on injury cases show the following remarkable facts, in addition to a far greater number of cases adjusted to conditions with handicaps less easily classified and enumerated than these graphic surgical disabilities.

No. employees on S. P. lines permanently injured, settled with and retained in service Oct. 1, 1926.

No. permanently injured employes			407
Position since injury	better	107	
	same or similar	175	
	less important	125	
			407
Salaries since injury	larger	233	
	same	99	
	less	75	
			407
Average No. years employed since injury			11
Age at time of injury—			
Under 30	118	at present	30
30 - 40	149	at present	76
40 - 50	101	at present	145
50 - 60	34	at present	111
60 - 70	5	at present	45
	407		407
Injuries—			
Amputations—arms			33
hands			11
fingers			75
legs			84
feet			14
toes			17
Fractures — skull			4
back			7
hips			7
arms			10
fingers			2
legs			24
ankles			4
feet			11
Sight lost — one eye			40
impaired — one eye			13
impaired — both eyes			3
Paralysis			1
Other Injuries			44

Were it not for the attitude of the Labor Unions, which prevent a member of one being transferred to any other service without entering a new union and sacrificing all of his seniority rights, the dealing with handicapped men in industry, whether the handicap be from accident or illness, would be simple. The Unions are his worst enemies and not his protection, when once it is desirable for him to cease the kind of work for which he is trained and enter some other field. One of the greatest needs in industry is for some reciprocity agreement among tbe Unions themselves in these cases.

It is evident that everything has been done by the Company through its supervisors, foremen, and hospital department, to convince the employees that, in sickness or injury, the interests of the Company, the doctor and the patient are precisely the same —that the man should be restored to full working capacity on his job, at the earliest possible moment. If the compensation laws could be framed so as to assist this situation, a good deal that is both difficult and disagreeable would be eliminated. For thirty years, the studies in Germany, Italy, Austria, Switzerland, France and England, particularly, have shown that, where the courts

recognize a covetous insurance or compensation neurosis and the unstable basis of the nervous systems of such cases, compensation, if awarded, is for the injury but not for purely neurotic symptoms. In other words, the law and the courts have the power of preventing this disorder, just as they alone can cure it by finally awarding compensation or throwing the cases out. A far better understanding of the conditions attending accident, shock, concussion, must be disseminated generally before we can hope for improvement in preventing and handling these cases. The problem is difficult because, frequently, absolute diagnosis is impossible. The true malingerer can sometimes present his subjective symptoms very convincingly. Hysteria provoked by injury and bad handling can simulate organic disease, but careful and thorough study of the patient—his medical history, his personality, reactions to varying problems, his present fundamental viewpoint and effort expenditure, as compared to what can be learned of him before the accident,—and finally, such a study and interpretation of symptoms, subjective and objective, as he presents, with all the available laboratory data, will give a pretty fair and dependable picture of the man and what his injury did to him. Of course, there will always be the human equation that presents itself as in the horse trade. The buyer points out and dwells on the shortcomings or defects and the seller confines himself to the good points.

It is evident that the covetous neurosis did not develop in these injury cases shown on the chart or, having developed, it was cured by compensation adequate to suppress it to within normal range.

Dr. Thomas Salmon, head of the psychopathic department of the A. E. F., covers the traumatic and covetous neurosis—the so-called shell-shocked cases—with these words: "The further back from the front line trenches such a case is allowed to get, the harder it is to get him back." If he can be handled adequately at once, he is adjusted to the situation, reconciled to what happened, possibly his more or less narrow escape, and all goes on well. If the soldier never wanted to go to war, did not like his commanding officer, was a natural coward, got word from home that his wife was not well or that a neighbor was paying a good deal of attention to the girl he left behind him, it is not difficult to realize how much time that man spent in being sorry for himself and how easily he might get the impulse to slump into a demoralized state if a shell happened at that moment to land even half a mile away. Once on the way to the rear, the importance of the shell-shocked state grew and its definite relation to release from a condition toward which he never was reconciled, became more and more fixed.

It must not be forgotten that many of these cases, arising both within and outside of industry, do sustain multiple injuries, for which the disability in degree and duration can be calculated and the fixing of compensation is relatively simple. The loss of an eye or the breaking of a leg is relatively measurable handicap, but purely subjective symptoms like headache, back pain, nausea, irregular and painful menstruation, are not comparable in any two cases nor measurable. It is very evident that the covetous idea develops after the accident, not as a direct result of it, but of the conditions which follow it. One does not sue an irresponsible, penniless peddler even if his broken-down antiquated truck runs into one's car after breaking all the rules of driving. The very irresponsibility of hundreds of drivers is the chief excuse for a compulsory insurance law. If we get it, the increase in suits may be enormous but justice will, possibly, be more evenly distributed. The man who pockets his loss now and says nothing, will get a fairer deal. The man who sees in the accident a chance to get a new car, has developed in him the "something for nothing" idea and will insist on compensation even if he has to work himself into a feeling that he is a real sufferer and unfairly treated. It is not hard for some people, for there are some who are born with no love for work, who are unstable nervously and have no ability to think out problems. Unfortunately, there are plenty of lawyers and doctors to help them play their game, which is to dwell on the conditions which are claimed as a result of accident until they can be presented in court with a demand for compensation. The question arises whether a statute should be passed making accident cases come up for settlement within a short time after occurrence. The practice of many lawyers and insurance companies of dragging out cases by repeated postponements until even the patient is sick of delay and ready to settle, is a most pernicious one, for the fight, when it comes, is a bitter one and many times cases that were once simple become quite complex. A trivial accident to a man who had six children could have been settled for $150, but a big corporation kept the man coming to their legal department for months. Finally he got pneumonia and died and the widow collected $6000. If an injured man exaggerates or hangs on to the original symp-

toms of his accident with covetous ideas, he is a better and better actor and a more skilful tool for the ambulance-chasing lawyer.

The real faults of our present system are the things that cause delay in settling cases and the inaccurate diagnoses and failure to study and define accident cases fully and with great care in their beginning.

The historical aspect of all these traumatic neuroses, of which the covetous neurosis is merely one, dates from the light thrown upon the subject by Hoffmann, in 1890, and the writings of Strumpell, in 1891, and Charcot, in France, earlier than this. Schultze and Strumpell together, in 1895, made a very decided advance in suggesting the word "Begehrungsvorstellung," which, being translated, means covetous imaginative conception.

Charcot stated, all of 40 years ago, that the German compensation law was responsible for accident neurosis. There is cited one striking example in German literature, of a railroad accident in Mullheim, in which over 400 soldiers were victims. There were no traumatic neuroses among them and all were well in a few months. In a similar accident occurring where compensation was possible, 99 were injured, of whom 52 became neurotic. His, of Berlin, quotes Ciampollini, an Italian railroad doctor, as having reported 160 neurotic patients, of whom all but four were able to return to work after having received the full amount of compensation. Bonhoeffer, in 1911, refers to the discussion in Germany after the International Congress in Berlin, in 1890, and shows how hard the opinion died that organic diseases underlie these neuroses. He calls attention to the growing idea, which again in 1896 he reiterates, that simple shock does not produce prolonged neurotic, hysterical, and psychic phenomena. The entrance of the covetous factor, alone, leads to the fixing of the symptom complex that is cured finally by the compensation. The world war produced an enormous amount of mass evidence that shock emotion symptoms were confined to soldiers at the front on both sides, but were not found among the prisoners who were out of danger from the war. Hospitals for neurotics were a necessity for soldiers in the trenches, but never for prisoners.

The older conception, that the injury produced permanent neurotic symptoms, was clung to in Germany as late as 1916, when Oppenheim, the last defender, accepted the ideas that now hold, that the brain and the nervous system are not vulnerable, as originally supposed. In spite of the far better understanding of serious injuries to the brain, it has stood out quite plainly that epilepsy and insanities, as evidence of permanent change, do not result in any greater degree because of injury. The real traumatic syndrome of brain injury or shock produces a retrograde amnesia and certain epileptoid symptoms in cramp and spasm attacks, periodic depressions, greater susceptibility to alcohol and other intoxications, excitability, vertigo, periodic headaches. This is, on the whole, a very different picture from the chronic covetous neuroses, which show tremor, vasomotor disturbances and inability to function perfectly with voice and limb, and a type of pain not to be defined in any organic diseases.

With our better diagnoses, we are not going to make the mistakes that will operate against the interests of patients, that have been made in the past, and this applies equally to regarding as cureable those cases which are organic.

Our compensation laws should be so framed as to rule out all chance of accident victims breaking the tenth commandment and they should be settled with, for whatever demonstrable accident effects were produced, and returned to duty at the earliest possible moment.

SUMMARY

If the tenth commandment were enforceable, two per cent of all accidents complicated by so-called traumatic neuroses would not have features involving legal and medical questions.

The present law is bad, for it encocrages covetous desires and neurotic reactions to minor conditions. The worst feature is the monthly payment plan, which assists in emphasizing the importance of the symptoms.

The long-continued practice employed by the Southern Pacific Company, of settling promptly with an injured man and getting him back on the job as soon as possible, minimizes all forms of neurosis—whether marked by concussion symptoms, shock or hysterical manifestations—reduces to a minimum the covetous influences which serve to exaggerate and fix the symptoms.

The pernicious practice of insurance companies and some lawyers, of delay and the use of every means of making suits unbearably burdensome to the plaintiffs in these cases, only serves to exert a bad influence on the economic, medical, and moral aspect of these cases.

The war experience showed that prisoners in hospitals suffered from no traumatic or covetous neuroses. However unstable nervously they might be, the fact that there was nothing to be gained exerted a remarkably beneficial influence. Fear, doubt and uncertainty no longer exerted their power.

DYSPNEA AND ORTHOPNEA WITHOUT DISCOVERABLE DROPSY

(Discussion of Case 14381, Case Records of the Massachusetts General Hospital, New Eng. Med. Jour., Nov. 8, 1928, p. 946).

CASE RECORD

A Scotch-American printer fifty-two years old entered the hospital December 8 complaining of a collapse four days before admission, with dyspnea, orthopnea and weakness.

Three years before admission he had occasional attacks of pain across the base of the heart lasting a few minutes, sometimes running up to the neck or through to the back. He was slightly dyspneic and tired more easily than usual. He was, however, in good health until a year and a half before admission. Then he had substernal oppression on exertion radiating to both arms, lasting a few minutes and relieved by resting. A year before admission all his teeth were extracted at once. This nearly finished him. Ten months before admission he had attacks of dyspnea, orthopnea and choking at night, often waking him. These increased until six months before admission, when he gave up work and had to spend nights sitting up in a chair. He was then seen by Dr. Paul D. White, who says that he was exhausted, had lost forty pounds recently and was very much discouraged. He had been taking fifteen drops of digitalis t.i.d. without relief. Examination at this time was similar to that done in the Out-Patient Department as reported below. He was referred to another physician of the hospital staff, who prescribed three months in bed.

Examination in the Out-Patient Department May 13, seven months before admission, showed a healthy looking man. The heart was enlarged to the left, with the apex in the sixth space. There was a precordial systolic and an early diastolic, more marked in the aortic area. There was wide percussion over the great vessels. Corrigan pulse. Blood pressure 202/82.

Three and a half months before admission the patient resumed very mild activity with some dyspnea and orthopnea.

November 10 he was sent to the South Medical Department. Examination showed his pupils and knee-jerks normal. A Wassermann was moderately positive. He was given a course of treatment. His general condition improved.

November 17 examination showed him pale and thin, but looking better than when seen by Dr. White six months earlier. The apex impulse of the heart was in the the sixth space, 11 centimeters to the left, 3 centimeters beyond the midclavicular line. The dullness in the second space was 5 centimeters to the right and 3.5 to the left. There was no abnormal pulsation at the base and no visible tumor. Normal rhythm. Rate 100. Blowing systolic and diastolic murmurs heard all over the precordia. Blood pressure 174/108; practically the same in both arms. Pulses not especially of the Corrigan type. Arterial pulse visible in the neck. Slight tracheal tug. Heart sounds largely masked at the base by murmurs. At the apex in the recumbent position was also heard a slight mid-diastolic rumble. X-ray examination at this time showed moderate increase in the transverse diameter of the heart, the greatest enlargement being in the region of the left ventricle. The supracardiac shadow was wide both in the anteroposterior and the oblique view. There was marked prominence of the ascending aorta, also in the region of the aortic knob. The oblique view showed marked dilation of the supracardiac shadow. Measurements; right 4.4, left 10, total transverse 14.4, great vessels 8, chest 24.5.

Four nights before admission he collapsed, sweat profusely and had to be taken home. Since that time he had been in bed, with paroxysmal dyspnea, orthopnea, very bad heart sounds and a short systolic and an aortic diastolic murmur. The morning of admission he was seen by his physician, who thought he was in extremis.

There is no family history except that a sister died suddenly of unknown cause.

No past history was obtained.

The temperature, pulse, respirations, urine and blood are not recorded.

The patient entered the Emergency Ward in complete collapse, ashy pale, cyanotic, sweating and gasping for breath. He was seen immediately and was sent at once to the medical ward, where it was hoped that a Gatch bed and rest would revive him. He died in the elevator.

Discussions by Group II, of the Yavapai County Medical Society and the Medical Officers of Fort Whipple, at their meeting of March 19, 1929.

DR. W. E. McWHIRT, Whipple, Ariz.

We are to consider a printer aged 52 who, as far as information given us is concerned, is seeking relief from dyspnea. Up until three years ago he had no particular trouble. At that time he began to have slight dyspnea on exertion, with some pain radiating over the base of the heart. He went along like this until about seven months ago, when he began to have considerable trouble and had to sit up at night. He could not lie down to sleep—if he did go to sleep the pain awakened him. Before this he had all his teeth removed, which nearly cheated us out of an interesting case. We presume he had an infection, pyorrhea or what-not.

At the examination in the outpatient department in May, he was not considered an overly sick looking man but he had lost about forty pounds; had an interesting blood pressure, which I think will carry considerable weight. He had a diastolic of 202 with systolic of 82 which means a pulse pressure of about 120. They find he has a positive Wassermann and our group attaches considerable importance to this, although we have been knocked off the pedestal several times by Dr. Cabot. However, we have in our favor the statement of a famous cardiologist, who says that, in all cases of aortic regurgitation in men over 42 years of age, the cause is syphilis until proven otherwise. At a later examination, after treatment, his blood pressure has changed to 174/108, which is quite a little change. The x-ray film gives us quite a bit of information. This point I want to stress. They give the measurement complete with the heart as 14.4, which is not very much and the chest measurement 24.5. He must have been a very small man, as the usual chest measures 28 to 30. They tell us that the supracardiac area is widened in both the lateral and oblique views. There are about four things which might give this widened shadow: aortitis, aneurism, tumor and abscess. We do not believe this is a history of abscess; it is impossible to rule out tumor completely, although, if tumor were present, we think they would have told us there was a cloudy area between the supracardiac area and the spine. We are inclined to think this shadow was caused by an aneurism in the region of the great vessels.

He goes on until four days ago when he has a complete sinking spell and is carried home in more or less shock. His physician saw him the day before

admission to hospital and thought him in extremis. He was taken to the hospital—they tried to send him to a ward, but he was in profound shock and died in the elevator.

We believe, with the lack of any history, no examination of the urine or blood, with the findings given, that we can limit this case to the heart. We are not justified in bringing the kidney into the case because there is nothing remarkable except his high bood pressure, which could very easily be caused by certain heart lesions.

DR. G. O. BASSETT, Whipple, Ariz.

Khel has stated that the human heart is the most perfect motor known to man. At the rate of sixty pulsations a minute—86,400 in twenty-four hours—the human heart does considerable work. It is a pendulum in constant motion, and despite all defects the body may have, it, in the majority of cases, maintains its work with less variation than any other organ of the body.

We have an interesting case here, which deals with the most interesting of all organs and with a heart lesion that is most interesting of all to contend with—a heart lesion in which the signs are supposed to be most classical when they are present, but they are not always present.

I shall attempt very roughly to touch on two types: first, the various aspects of aortic regurgitation, a heart lesion with characteristic symptoms and one in which it is very difficult to get results by treatment. All cases, of whatever cause, start as this case has started, with dyspnea, pain across the base of the heart running into the arms. This is usually the first and only symptom of which the patient complains. The first stage indicates simply that we have a heart lesion, probably aortic regurgitation. If we examine the patient at that time, nothing else of importance will be found—the heart not enlarged materially, if at all, the murmur present will be very soft, short diastolic, or it may be absent. So that at this stage the best advice, the authorities agree, is to continue on and avoid exertion. When the second stage is reached there is more marked dyspnea, orthopnea, and choking spells, especially at night. At this stage there is early failing compensation, where the heart has already hypertrophied to a certain extent, is possibly dilating as a result of too much strain. The patient is now reaching a stage of danger and must be warned against, not only extra exertion, but any exertion. There is one point I should like to bring out. The history tells us that Dr. White called on this patient and found he was receiving fifteen drops of digitalis t.i.d., without relief. In most cases of heart lesions, the first medication thought of is digitalis. Aortic regurgitation, however, is a condition where digitalis is poison and acts, as you can see, by doing the exact opposite of what is wanted: by prolonging the systole it allows more blood to flow back through the leaky valve and puts more strain on the left ventricle, which is already overstrained.

Later, this patient came to the dispensary, was examined and found now to have definite evidence of aortic regurgitation. There is present a diastolic murmur, which is typical—soft, short. In addition to that he has a systolic murmur, which we are told is classical in these cases and due to the cross current of blood mingling in the left ventricle, blood flowing back through the leaky valve and blood flowing out.

This man, we see later, receives treatment in the venereal section of the hospital and improves. However, a man with aortic regurgitation of any extent could not be expected to overcome that difficulty, even with salvarsan, and although his general condition improved, we note that, by the next examination, the actual lesion has apparently increased. Another point before I go on. It was hard to understand why a man without arteriosclerosis—and a great many of these cases do not have it—could have a blood pressure of 202. The answer was very simple and the explanation, we believe, fits this case. The high systolic pressure is due to the terrific functional effort of the left ventricle and the sudden drop to a low diastolic pressure, due to regurgitation through that leaky valve. This man's pulse pressure we see is terrifically high and is due to the extra effort of the heart muscle, raising thereby the systolic and causing the marked disproportion in the diastolic. We see then that, even if that tremendous pulse pressure is present, it is not necessary to have arteriosclerosis or change to any extent in the arteries, particularly the aorta. Later we find this blood pressure dropping; it drops something like thirty points systolic and the diastolic goes up. Our interpretation of that is that it is due simply to a failing myocardium. The myocardium has become so weakened it is unable to withstand that terrific jolt and the systolic drops from increase of aortic regurgitation.

This man has most of the classical symptoms. He has a diastolic and systolic murmur; he has evidence of enlargment of the heart, and has, when first examined, Corrigan pulse. Later, they tell us, he has not an especially typical Corrigan pulse. That it was present once and then disappeared is due to the fact that we are dealing with two conditions. We believe this man has an aneurism of the ascending arch of the aorta—aneurism cutting off the terrific impulse that goes with that so-called "water hammer pulse"—as well as aortic regurgitation.

DR. JOHN W. FLINN, Whipple, Ariz.

Aortic regurgitation, or aortic incompetency, is a pathological condition of the heart in which part of the blood that has been pumped from the left auricle into the aorta, is regurgitated back into the left ventricle, because the semilunar valves are incompetent to close the aortic opening. If one visualizes a condition such as this, it will at once occur to him that this may be caused by one of two conditions. This incompetency may be caused by a dilated aortic ring, or it may be caused by a deformity of the semilunar valves. So there are three commoner causes of aortic insufficiency, two of them are associated with the deformity of the valve, and one of them is the dilated aortic ring. These three causes are:

1. Progressive sclerosis of aortic valves, being part of a general arterial degeneration, or it may be due to a localized syphilitic lesion.
2. Definite endocarditis, which may be either rheumatic or malignant.
3. Dilatation or aneurism of the ascending arch of the aorta. Of these three causes, the dilated ring occurs in a fairly large percentage of cases of aortic incompetency. Whatever the cause, this incompetency, with its accompanying regurgitation, gives rise to fairly definite signs and symptoms, which are largely the same regardless of the cause.

Group two believes that there is almost certainly an aortic incompetency in this case. We cannot see how there could be so many of the classical signs and symptoms unless these valves are incompetent. The question which bothers us greatly it, "What is the cause of this incompetency?"

You will remember that there are five signs of aortic incompetency, which have been given by

previous speakers: (1) A diastolic murmur heard over the aortic area and produced by the reflux of blood into the ventricle; (2) the so-called "Flint" murmur, a harsh mid-diastolic murmur, which is heard in almost fifty per cent of cases; it is supposed to be due to the impinging of the regurgitant current on the large anterior flap of the mitral valve; (3) a collapsing Corrigan pulse brought about by the fall of pressure during diastole; (4) enlargment of the heart and especially of the left ventricle; this is due at first to a compensating hypertrophy and later to a decompensating dilatation; (5) visible pulsation in the peripheral vessels, especially those of the neck. All these signs and symptoms are present in this case.

Aortic insufficiency, in common with other forms of heart disease, gives rise to a fairly definite train of symptoms.

The x-ray findings are fairly suggestive of aortitits. Almost surely we have a dilated aorta, whether or not it is simply an enlargement or whether it may be an aneurism, the group is not prepared on this point to commit itself. The fluorscope was not used in this case and, consequently, we do not know that we have a pulsating tumor. It is true we have tracheal tugging but we are told this may be present in pure aortic incompetency. The soft systolic murmur may be aneuryismal but it is found also in aortic incompetency, or it may be due to an accompanying aortic stenosis. This patient's heart is undoubtedly greatly dilated, and is probably leaking at all the valves, which would account for all kinds of murmurs all over the heart.

From these two standpoints we are prepared to make a definite diagnosis of aortic incompetency and a definite diagnosis of aortitis. We are not, however, prepared to commit ourselves that there is a definite aneuryism present.

Regarding the mode of death, that, too, is inconclusive. This patient died suddenly, but so far as we know there is nothing absolutely conclusive about this form of death. If we think of this case as a whole—a man with a cardiac lesion, with a dilated aorta, and a positive Wassermann (for which he evidently received treatment)—we could very well assume coronary thrombosis; or if we have an aneuryism, it could very well have caused this sudden death by bursting into the pericardium. The question is, have we these signs, will the autopsy reveal plaques on the aorta? Will it reveal a sclerotic condition of the coronary vessels and signs of luetic degeneration in the heart muscle? We don't know. So, we are driven back to our former diagnosis of aortic insufficiency and aortitis.

We are willing to speculate regarding the other details of the pathologic picture. We are quite willing to admit that all of these other conditions I have mentioned may be present and that some of them probably are, but we do not feel justified in concluding definitely that they are present. Our diagnosis is aortic incompetency and chronic aortitis.

Discussions at Massachusetts General Hospital.

RICHARD C. CABOT, M. D.

We can sum up the present illness by saying that a man of fifty-two has shown two manifestations of heart trouble, passive congestion and angina pectoris. He has dyspnea, orthopnea and pain. All these seem clean-cut enough. There is nothing else complained of to make us look elsewhere in the body. We have also this aortic diastolic murmur, which is very important if confirmed. The short systolic does not help us much.

He was given a course of treatment in the South Medical Department. That in itself is evidence of syphilis. They not only found his Wassermann positive, but (since they treated him) they thought he had syphilis, which is a different thing. They never begin treatment for a Wassermann alone. So the Syphilis Department, who know more about it than the rest of us, think he had syphilis.

"His general condition improved." That is surprising. We do not expect that under treatment for syphilis if there is syphilitic aortitis. We always give that treatment because it is the only rational thing to do, but I am surprised when they improve at all.

NOTES ON THE PHYSICAL EXAMINATION

It is always satisfactory to have a definite confirmation of a Corrigan pulse as shown in the pulse pressure of 120. We have every reason to be confident that they were right in what they said about that aortic diastolic murmur. We are thinking then of the type of heart disease with great cardiac hypertrophy and with aortic regurgitation, apparently not of one of the main types, syphilitic or rheumatic. We have nothing in the history to make us think of either of those things. But we shall look for evidence of them as we go on.

That heart certainly is enlarged if we pay attention to percussion. One is thinking of a definite disease in the aorta. 8.5 is certainly too wide.

They have done what they could to find aneurysm. "Blood pressure 174/108; practically the same in both arms." That is not so clear as it was before in favor of what we call Corrigan pulse.

"Slight tracheal tug." What does that mean?

A student: Aneurysm.

Dr. Cabot: Yes; but you can go wrong on tracheal tug in two important ways. First a pulsation that goes in and out, forward and backward. Put your finger under the cricoid and you feel that pulsation. That is nothing. Tracheal tug must be an up and down pulsation. The second source of error is a tracheal tug that comes during inspiration only. A good many patients without aneurysm have this. In my experience it is very rare to be sure of it. It must be up and down and it must go with every beat continuously. It is a very important sign if it is present. There is almost no record of its occurrence in anything else but aneurysm.

That mid-diastolic rumble tends to swing us over to the other type of heart disease, rheumatic; but otherwise all the evidence is the other way.

The radiologists do not definitely say whether they think there is aneurysm or not. It is my impression that they do, at any rate that the arch is abnormally enlarged. That was their statement, that is was a diffuse dilation, but they could not find any single aneurysm fluoroscopically. Of course we have to remember that mere arteriosclerosis can give us diffuse aortic dilation without any aneurysm at all. Aneurysm breaks and arteriosclerotic dilation does not—a very important difference for the patient. Otherwise you might say that it is merely a play on words to distinguish between dilatation and aneurysm. Diffuse arteriosclerotic dilatation does not give pressure signs, does not cause pain, does not break. It is very important to make up our minds which it is. But I cannot be at all sure from that x-ray examination. It may turn out not to be aneurysm when the secrets of the facts are revealed, but I should say that that could not be an ordinary arteriosclerotic aorta.

DIFFERENTIAL DIAGNOSIS

Let us start with the things we know best and work out towards our uncertainties. We know that

he has aortic regurgitation. We do not know of any other valve lesion. He is a middle aged man or past it, and it is a good general rule that a man who begins in middle age to suffer from heart trouble, and that aortic regurgitation, has syphilitic aortitis. In support of that is the pain, which we often see in aortic regurgitation due to syphilitic degeneration, because the process goes around the mouths of the coronary arteries. We do not get such pain in rheumatic aortic trouble. The patient's pain inclines us still further to believe this is syphilitic. Also we have a positive Wassermann and the diagnosis of the South Medical Department on which they must have started their treatment. At one time he has had a Corrigan pulse. Corrigan pulse is much commoner with syphilitic disease in the aortic valve than it is with rheumatic disease in the aortic valve. Rheumatic trouble in the aortic valve makes the leaflets stick together, not in every case but in at least four out of five, and therefore stenosis goes with it, and with stenosis you rarely get a Corrigan pulse. On the other hand syphilis draws back the leaflets, widens them, and gives you aortic regurgitation such as goes with Corrigan pulse. That evidence is weakened by the fact that on second examination he did not have anything that was suggestive of Corrigan.

A student: Dr. White's opinion was that it was not Corrigan.

Dr. Cabot: Dr. White says it is not?

A student: That is, he said it was only suggestive, not definite.

A student: What evidence have we of passive congestion?

Dr. Cabot: Very little is said about it. I should like to know what they found in his lungs and legs. If he had no passive congestion we should look for a type of disease blocking the coronary rather than one causing stasis.

A student: How do you explain the dyspnea and orthopnea otherwise?

Dr. Cabot: It is hard to. You have to say that there is some passive congestion without clear signs. But such a patient ordinarily shows some fluid. Dropsy comes a little later than difficulty in breathing.

Dr. Tracy B. Mallory: It is not in the record, but Dr. William D. Smith saw him the morning of admission and said that at that time, although he did not want to examine him very much, he found no dullness at the bases, but a few rales.

Dr. Cabot: And nothing is said about his legs?

Dr. Mallory: No. No edema.

Dr. Cabot: In the three "L's"—lungs, liver and legs,—look especially for passive congestion. Dr. Mallory says no edema, so I should say there is remarkably little evidence of passive congestion, which strengthens us on the side of the syphilitic process, because a syphilitic process involving the coronaries may produce no stasis. On the whole I think, as I imagine you do, that this is syphilitic aortitis with aneurysm, with narrowing of the coronary arteries and death from the trouble rather than from a rupture of the aneurysm into the pericardium, which is the other common cause for sudden death in such a case as this. Aneurysm rarely breaks externally. Often they break into the pericardium. He may have had that, but we have no reason to suppose so.

In the rest of the body we have little to make us think of disease. Have we a urine examination?

Dr. Mallory: No.

Dr. Cabot: On the face of the facts given I do not see what more we can say.

A student: Do you ever get tracheal tug in aortic insufficiency without aneurysm?

Dr. Cabot: Not so far as I know. I never heard of it. The only thing besides aneurysm is a tumor in close apposition with the aorta.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Luetic heart disease.

DR. RICHARD C.CABOT'S DIAGNOSIS

Syphilitic aortitis with aneurysm.

Narrowing and occlusion of the coronary arteries.

ANATOMIC DIAGNOSES

1. Primary disease.

Syphilitic heart disease—aortic insufficiency.

Syphilitic aortitis.

Aneurysm of aortic arch with erosion of manubrium.

Coronary sclerosis.

Fibrous myocarditis.

Adhesive pericarditis.

2. Secondary or terminal lesions.

Arteriosclerotic nephritis.

Chronic passive congestion.

DR. MALLORY

The first thing we noticed in removing the sternum was that something was tightly adherent beneath it. It stripped off with difficulty and left this little shallow, hollowed-out depression in the posterior surface of the manubrium. Into this depression fitted the middle one and most sharply defined of the three aneurysmal dilatations. We have a diffuse big sac here at the base, then a sharply outlined one partially filled by thrombus, from far down inside of which the innominate artery takes its origin, and finally a third more diffuse dilatation of the descending thoracic aorta. From the diaphragm down the aorta was not remarkable.

The aortic valve shows surprisingly little involvement. Two of the cusps are perfectly normal. The third one shows at this point a cusp anchored down to the wall. There is not, however, any lesion of the commissure, which is the ordinary spot at which syphilis affects the valves. It unquestionably would have given him a slight degree of regurgitation, but not, I believe, a very marked one.

The type of hypertrophy of this left ventricle fits this assumption. In a hypertensive heart disease one gets a so-called concentric hypertrophy of the left ventricle, that is, it is widened and elongated in almost equal proportions. In a true aortic regurgitation with marked functional regurgitation one gets very marked lengthening in proportion to the widening, often spoken of as eccentric hypertrophy. This ventricle is much more of the concentric type. So we may assume that he had a slight regurgitation, but not a very marked one. The regurgitation itself probably did not have much to do with his death.

The myocardium shows a number of white fibrous scars. The reason for these is obvious on examination of the coronary vessels, which are calcified, their lumen almost entirely occluded. It is not, however, the syphilitic type of coronary disease. The mouths of the vessels are perfectly clear. The important syphilitic involvement seems to begin safely above the level of the valve. The picture in the coronary arteries is apparently a purely arteriosclerotic one. To go with that he had a very marked degree of arteriosclerotic nephritis of the small vascular type and finely granular kidneys, moderately shrunken. So that I think the arteriosclerosis must be placed very high in estimating the cause of death in this case. It is impossible to say whether it is primarily due to arteriosclerosis or to syphilis. It certainly is not purely syphilis, though the rather sudden end, of

course, is characteristic of syphilitic disease of the heart.

It is interesting that Dr. Smith, in going over his lungs four hours before death, found no dullness at either base. At the time of necropsy well over a liter of fluid was found in each side which must have accumulated with extreme rapidity during those last three hours. The lungs themselves did not show any marked edema. The accumulation was entirely in the pleura.

There was no ascites and no demonstrable edema of the legs at post mortem. The liver did show a moderate amount of chronic passive congestion and two scars which possibly may be syphilitic in origin.

Dr. Cabot: What would you guess the heart weighed if you had it separate?

Dr. Mallory: Between five and six hundred grams.

Dr. Cabot: You do not believe that aneurysms cause cardiac enlargement?

Dr. Mallory: No, sir. I think the kidney is probably chiefly responsible in this case.

Dr. Cabot: Did you say anything about the mitral valve?

Dr. Mallory: Entirely negative.

Dr. Cabot: I do not think they can blame us for not making the kidney diagnosis when they gave us no urine report.

The question is asked. "Are there cases of syphilitic aortitis without aneurysm?" There certainly are and in the majority, and with no regurgitation of the aortic valve and no involvement of the coronary arteries. The earlier and I suppose the commonest types of syphilitic aortitis do not give any one of these things. It is when it goes on for a long time that the disease gives a picture like this. Syphilitic aortitis in early stages simply means a ring of disease around the aorta above the valve.

A student: Do you mean it eventually goes on to aneurysm?

Dr. Cabot: No. Many of them do not. In France I saw a very large number of necropsies on Negro soldiers who had syphilitic aortitis. None of them had aneurysm and none of them had any of the other things that do any harm. They were perfectly well men until they were hit in battle.

NEW MEXICO ANNUAL MEETING

NOTES OF THE MEETING

The attendance was surprisingly large, but a double-header always draws a large crowd, so it was only natural that a State meeting at one of the most beautiful and picturesque places in the State and country, should be an all-star attraction.

Those registering were:

Dr. Dwight Allison, Las Cruces, N. M.
Dr. P. L. Atkins, McGaffey, N. M.
Dr. and Mrs. C. R. Bass, Cimmaron, N. M.
Dr. M. F. Bean, Albuquerque, N. M.
Dr. C. F. Beeson, Roswell, N. M.
Dr. W. C. Barton,
Dr. R. L. Bradley, Roswell, N. M.
Dr. Robert O. Brown, Santa Fe, N. M. and Mrs. Brown.
Dr. H. T. Brasell, Portales, N. M., and Mrs. Brasell.
Dr. W. L. Brown, El Paso, Texas.
Dr. and Mrs. M. O. Blakeslee, Las Lunas, N. M.
Dr. and Mrs. L. B. Cohenour, Albuquerque, N. M.
Dr. F. H. Crail, Las Vegas, N. M.
Dr. M. B. Culpepper, Carlsbad, N. M., and Miss Culpepper.
Dr. George H. Curfman, Salida, Colo.
Dr. C. B. Elliott, Raton, N. M.
Dr. and Mrs. Crum Epler, Pueblo, Colo.
Dr. T. Espinosa, Espanola, N. M.
Dr. J. Espinosa, Espanola, N. M.
Dr. Joseph Foster, Santa Fe, N. M.
Dr. Evelyn F. Frisbie, Albuquerque, N. M.
Dr. E. W. Fiske, Santa Fe, N. M.
Dr. F. C. Goodwin, El Paso, Texas.
Dr. and Mrs. M. S. Gregory, Oklahoma City, Okla.
Dr. C. H. Gellenthien, Valmora, N. M.
Dr. and Mrs. W. F. Glasier, Carlsbad, N. M.
Dr. R. J. Groom, Carlsbad, N. M.
Dr. and Mrs. P. R. Giliven, Santa Fe, N. M.
Dr. L. A. Hubbard, Raton, N. M., and Mrs. Hubbard.
Dr. and Mrs. E. T. Hensley, Portales, N. M.
Dr. and Mrs. C. F. Hegner and son, Denver, Colo.
Dr. J. W. Hannett, Albuquerque, N. M.
Dr. and Mrs. J. R. Hurley, Antonito, Colo.
Dr. Beryl R. Johnson, Topeka, Kans.
Dr. and Mrs. W. T. Joyner, Roswell, N. M..
Mary Frances Joyce, Carlsbad, N. M.
Dr. W. E. Kaser, Las Vegas, N. M.
Dr. H. A. LaMoire, Pueblo, Colo.
Dr. W. R. Lovelace, Albuquerque, N. M.
Dr. J. F. Lane, Santa Fe, N. M.
Dr. G. S. Luckett, Santa Fe, N. M.
Dr. W. J. Latta, Wagon Mound, N. M.
Dr. Gertrude Light, Ranchos, N. M.
Dr. K. D. Lynch, El Paso, Texas.
Dr. T. P. Martin, Taos, N. M.
Dr. and Mrs. Karl A. Menninger, Topeka, Kan.
Dr. W. P. McCrossin, Colorado Springs, Colo.
George A. McAlmon, El Paso, Texas.
Roberta Bradley Markus, Roswell, N. M.
Mary Catherine Massie, Roswell, N. M.
Dr. J. N. Medett, Jarvis, Colo.
Dr. H. P. Mera, Santa Fe, N. M.
Dr. Carl Mulky, Albuquerque, N. M.
Dr. Orval I. Nesbit, Espanola, N. M.
Dr. J. A. Reidy, Albuquerque, N. M.
Dr. W. E. Rice, Raton, N. M.
Dr. Leon Rosenwald, Kansas City, Mo.
Dr. W. H. Spitzer, Denver, Colo.
Dr. G. W. R. Smith, State College, N. M.
Dr. William Senger, Pueblo, Colo.
Dr. M. S. Smith, Raton, N. M.
Dr. and Mrs. D. D. Swearingen, Roswell, N. M.
Dr. G. A. Sheppard, Gallup, N. M.
Dr. W. E. Sistrunk, Rochester, Minn.
Dr. and Mrs. W. A. Thompson, Springer, N. M.
Dr. Louis N. Tate, Galesburg, Ill.
Dr. and Mrs. W. Warner Watkins and daughter, Phoenix, Arizona.
Dr. M. R. Warden, Albuquerque, N. M.
Dr. and Mrs. G. Werley, and son, El Paso, Texas.

The election of Dr. Robert O. Brown, Santa Fe, as President-Elect was a very popular one and it speaks very well for the Society to have at its head a member of the State Board of Health. Other officers elected were as follows:

Vice-President—Dr. J. A. Reidy, Albuquerque, New Mexico.

Secretary-Treasurer—Dr. L. B. Cohenour, Albuquerque, N. M., (re-elected).

Members of the Council for Three Years—

Dr. C. B. Elliott, Raton, N. M.
Dr. Harry Miera, Santa Fe, N. M.

Invitation was extended by the Colfax County Medical Society through its delegate Dr. C. B. Elliott, to hold the next annual meeting of the Society at Raton, which invitation was duly accepted, with request that the date of the meeting be set at not earlier than June 1st, so that warmer weather will prevail and overcoats will not be in vogue.

The dinner dance at the San Geronimo Lodge was a well attended function and throughly enjoyed by all. It was a welcome sight to see some of the older members hoofing it around with the elasticity and grace of those of the younger generation.

Members and visitors who had never seen the annual corn dance or other Indian dances were deeply impressed with the finished and artistic performances as presented in the lobby of the Don Fernando Hotel and at the Pueblo. Proceedings of the scientific session Thursday afternoon were hurried considerably to allow the members to be guests of the Taos Lions' Club at the Pueblo to see the annual Corn Dance, which arrangements had been perfected by Dr. Martin, of Taos. The entertainment was a novelty and one that can be seen only at the Indian Reservations, and at that the dances at the various Reservations are different, entirely so.

The visits to the Artists'Studios were a treat and delight to all. Some of the most noted artists in the world make Taos their headquarters and their exhibits were viewed with all the zest and gusto of the connoisseur.

Fishing in the trout-streams was a keen pleasure not overlooked, especially by the younger generation. Dr. Martin, who knows every pool, gave valuable and wise hints on where to go and the proper kind of bait, with the result that those who went returned with a happy smile, showing the captures made, and telling of the larger ones that got away.

New Mexico State Board of Public Welfare

DR. G. S. LUCKETT, Director

MENINGOCOCCUS MENINGITIS AND MEASURES FOR ITS CONTROL

Under the above title, Dr. G. W. McCoy, Director of the U. S. Public Health Service's Hygienic Laboratory, outlined our peresent knowledge on the subject in a recent paper read before the Twenty-seventh Annual Conference of State and Territorial Health Officers (Public Health Reports, July 5, 1929, V. 44, No. 27). The tone of the paper was necessarily pessimistic, but hope lies in the research work that goes on incessantly. Salient extracts from the paper are reproduced below.

"Apparently during the past year we have had in the United States a more severe outbreak of epidemic cerebrospinal meningitis than at any time since the World War, and, indeed, we must go back to about 1905 to find the last comparable prevalence of the disease. In general, the condition has been more serious in the West than in the East. Indeed, it may properly be said that in some western communities the prevalence of the disease has been distinctly alarming. There is no question that there have been outbreaks of very marked severity.

"So far as symptoms and diagnosis are concerned, nothing need be said here save that it appears to be not sufficiently recognized that cases may occur without characteristic symptoms referable to the central nervous system. Purpuric cases fatal in a day or less may occur even without examples of the classical type in the community. Diagnosis in such cases perhaps can be established definitely only by laboratory procedures, either the isolation of meningococci from the blood stream or the finding of the organism in smears or sections from the skin lesions.

"There have been generally recognized four types of meningococci based on the agglutination, or agglutinin absorption tests, which are in common use. There are other serological means of identification, and tests made by different procedures do not necessarily give identical results. One thing seems quite clear—that in any outbreak we may have in the same community, all or nearly all of the types prevailing simultaneously.....

"The question of types naturally brings me to the possible value of therapeutic sera. Generally speaking, we are accustomed to say that polyvalent serum has reduced the mortality from about 75 per cent to about 25 per cent. I am not familiar with any series of cases which definitely establishes the value of serum in terms that permit trustworthy statistical conclusions. It has been charged within recent years that the commercial serums have proven ineffective, and we have heard a great deal about the high mortality in certain series of cases in which commercial serums have been used. I do not doubt at all that the facts are as stated, but we have not heard so much about series in which commercial serums have been successfully used. For example, Carbonell and Cook, of the Army Medical Corps, report 19 cases with 2 deaths, making a mortality of less than 11 per cent. Commercial serums were used and, as you see, the death rate is very much lower than is claimed for serum treatment in general. Again, Wildman, of the Naval Medical Corps, reporting an outbreak in northern Haiti, had 77 cases with 12 deaths, a mortality of 15.5 per cent—also much better than the best that ordinarily is claimed for serum.

"Fox reported 12 deaths among 69 cases, excluding fulminating types which died within a few hours after admission to the hospital, also a very good result for the best of serums.

"There is hardly any more unsatisfactory standardization in connection with biological products than the present day standardization of antimeningococcus serum. The methods we employ may or may not have any bearing on the clinical usefulness of the serum. About the best we can hope to do (with our present knowledge) is to see to it that animals are immunized with a sufficient variety of specific type strains to insure reasonably high serological

titers for the generally recognized types of meningococci. We know from experience that such a serum will fail to meet the needs in some cases.

"The question has been raised as to whether it is possible to prepare a serum to meet the types prevailing in any given outbreak. The difficulties here are almost insuperable. . . . .

"We are endeavoring to meet the situation as respects the potency of the serum by having one of our best bacteriologists study the strains that have been secured recently, with a view to selecting those most suitable as antigens and utilizing them in the immunization of horses for the preparation of serum.

"I wish that we were in a position to tell you that as the result of scientific investigation we were in possession of definitely effective measures for the control of meningitis. However, nothing could be further from the truth than this, and I could perhaps dismiss this phase of the discussion by saying simply that we know nothing of any value. Possibly such a summary disposal of the subject is not warranted.

"The one measure on which everyone agrees is that persons suffering from the disease should be isolated. This seems to be a reasonable procedure and in keeping with the handling of other infectious diseases.

"The question of disinfection of carriers has been the subject of a great deal of discussion and of some experimentation. Many chemical disinfectants have been employed as solutions, sprays, or in the form of vapors. I am not acquainted with any data that would be seriously considered as conclusive which illustrate the value of any of these preparations, and Colonel Vaughn says:

" 'Notwithstanding the many experiments resorted to for this purpose nothing definite has come out of them, and from a fairly large experience and observation with these procedures during the war we are inclined to the conclusion that there was no demonstration of their value. * * * There is one thing that clears up the carrier, and that is life in uncontaminated, not overheated, air.'

"Most carriers clear up within a few days regardless of what one does or does not do, and it remains to be shown that the process of recovery from the carrier condition can be hastened by any means now at our disposal.

"During the prevalence of infection, carriers are very numerous. In a study made in Detroit recently it was reported that 46 per cent of household contacts were found to be carriers, while as high as 6 per cent of unexposed persons showed meningococci in the nasopharynx. Data of this sort lead one to realize the well-nigh hopeless nature of any attempt to control the carrier.

"Perhaps I might close this section on the control of epidemic meningitis by a quotation from Rosenau:

" 'It is not clear that any of the measures so far taken have either materially influenced the course of epidemics or prevented the spread of the disease.' "

MALARIA SITUATION IN DONA ANA COUNTY IMPROVES

Dr. C. W. Gerber, full-time Health Officer of Dona Ana County (N. M.) has summarized the results of intensive malaria control activities conducted in his county during the last 18 months.

As summer advanced, intensive irrigation produced many stagnant pools of water. At the same time, vegetation again filled the drainage ditches that had been cleaned last year. This produced an ideal situation for the further breeding and harboring of mosquitoes. But the actual condition prevailing at this time is distinctly encouraging.

It is his practice to take regular "dippings" from the pools and canals, counting the number of larvae found in each "dip" and averaging for each station where observations are taken. Following this procedure during the spring, he finds "a very slight increase in the number of larvae. But practically all collected are not beyond the second instar of development. In only two places were pupae discovered, one a frankly missed place and the other an undiscovered siphon". All of these larvae proved to be of the **pseudopunctipennis** variety, even after breeding them out to maturity. This variety has little effect in the transmission of malaria. All adult mosquitoes caught were also of this same variety. He says further that all areas are reported by local observers to be so free from mosquitoes that many persons are sleeping outdoors unprotected by netting, something that has not been possible in years.

A comparison of an untreated area with the section described above shows that breeding of mosquitoes is continuing there unabated.

The principal methods of mosquito control that have been applied are Paris green dusting of pools and water courses and the planting of top minnows (Gambusia). Dr. Gerber gives the minnows some credit for results obtained. The dusting operations are the most extensive of any in the world.

Last year, 438 cases of malaria were reported from this county, some of them probably relapses. Through June of this year only four cases have appeared that were definitely new infections and probably two of these were acquired last fall. It is encouraging to note that an increasing number of persons are using the "Standard Treatment" and that many of these are showing negative blood specimens. Over 1400 blood smears have been examined in the last twelve months.

A house-to-house survey was made, last year, and infected persons were given a sheet of instructions on prevention and cure. This year the survey will be repeated.

Most gratifying of all are the reports from merchants and farmers that general sickness incidence and labor turn over have been low.

TAT GETS RESULTS IN MORA COUNTY

Dr. J. J. Johnson, Health Officer of Mora County (N. M.), reports that he has personally administered 1362 complete toxin-antitoxin treatments during the last twelve months. This is part of a campaign that he has been conducting for more than a year. As a result, only two cases of diphtheria were reported to the Health Officer, during the year, a woman of sixty and a baby 2 years old. The previous year, 28 cases were reported.

NEW MANUAL ISSUED

The New Mexico State Bureau of Health has prepared and issued a Manual of Instructions for subregistrars, giving in the simplest possible form the gist of registration laws and procedure. A copy has been sent to each of the 350 subregistrars and it will be sent to anyone else who can make good use of it.

THE EMPYEMA PROBLEM
With Particular Reference to Proper Drainage*

JOHN ALEXANDER, M. D., F. A. C. S.
Ann Arbor, Michigan

(Annual Address on Surgery before the Arizona State Medical Association, Prescott, Arizona, April 18, 1929.)

In the June issue of this journal, the illustrations of this article were confused, Figures 1 and 4 being transposed. The following is the proper order for the illustrations, with the respective captions:

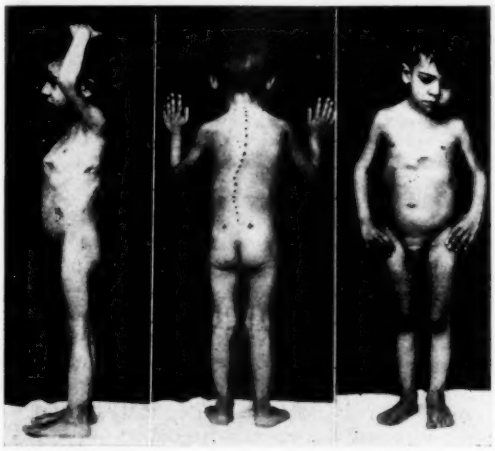

Fig. 1. Shocking deformity in a neglected case of empyema. Appearance on recent admission to University Hospital; febrile and almost constantly coughing and expectorating pus from empyemic cavity via broncho-pleural fistula. Pneumonic onset two and a half years ago. Complicating empyema not drained until four months later and then incision above bottom of cavity and tube removal after a short time. Resulting thoracic wall sinus has alternately opened and closed ever since, at no time furnishing adequate drainage.

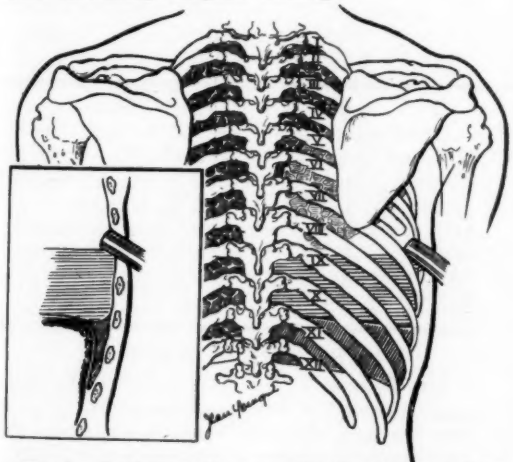

Fig 2. Drainage tube wrongly placed not at lowest limit of pus in empyemic cavity. Failure to provide second tube for antiseptic irrigations. Only pus above level of tube has drained off. Lung that pus had pushed away from ribs, as well as upward, is now visible but still is collapsed. Inset represents a frontal section of main drawing. In Figs. 2, 3 and 4 adhesions obliterating the costophrenic sinus are represented by oblique lines; pus by horizontal lines; fully expanded lung by dark lobules and collapsed lung by light lobules.

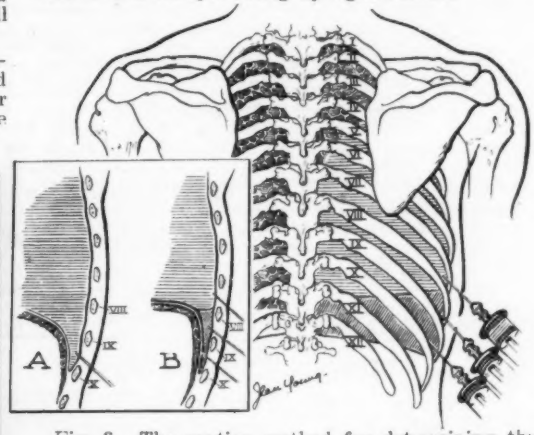

Fig. 3. The routine method for determining the site for dependent drainage in the posterior axillary line is to introduce an aspirating needle in the ninth intercostal space, and then in successive higher intercostal spaces, until pus is found. In the early stage of an acute empyema, pus would be found in the ninth space, which is just above the bottom of the normal pleural cavity (inset A). Several days after the onset of an empyema, adhesions bridge the narrow costophrenic sinus and raise the floor of the pus-containing cavity so that the aspirating needle would fail to find pus in the ninth space, and perhaps also in the eighth, but would find it in the seventh. This is illustrated by the main drawing, of which inset B is a frontal section. Therefore, dependent drainage would be attained by resection of a portion of the eighth rib (should open drainage be indicated). See Fig. 4.

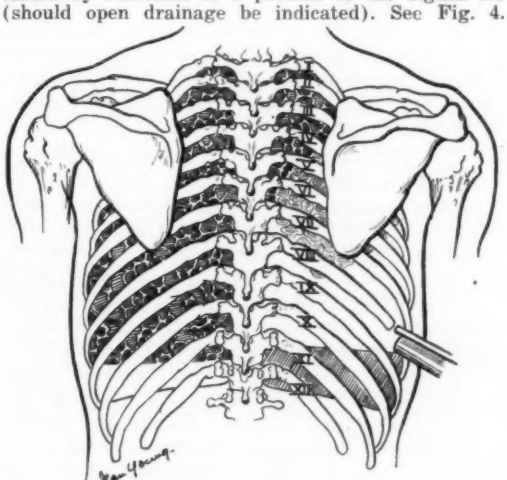

Fig. 4. Proper drainage at very bottom of cavity. All pus has drained away. A second tube has been provided for antiseptic irrigations. Steady expansion of the collapsed lung to obliterate the cavity will not now be checked by pus pressing against the lung, nor by the restraining influence of active infection in the pleural walls.

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EPIDEMIC MENINGITIS

A resume of an article by Dr. G. W. McCoy, Director of the Hygienic Laboratory, will be found in the New Mexico State Bureau of Health's section, elsewhere in this journal. This article brings to our attention certain well known facts about epidemic meningitis, but though well established, they are often lost sight of. Among the facts that the general practitioner frequently over looks are these:

1. Carriers spread the disease, and some unknown carrier is a more potent source of danger for other people than is a patient who already has the disease.

2. The strium of infection is the respiratory tract, from which the organisms invade the blood stream. The organisms inhabit the respiratory tract of many people, as does the pneumococcus, invading the blood stream and causing infection only after the general physical resistance is lowered.

3. Meningitis is a terminal stage of meningococcus infection. In the outset it is a bacteremia, and the patient may succumb to the infection before the meninges are involved. In those cases where the patients develop purpuric spots of varying sizes and die within a few hours (spotted fever), meningitis may not be present at all and the spinal fluid may be clear. In at least one instance in Phoenix, in the stage of purpura, blood culture recovered the meningococcus from the blood stream, the spinal fluid was clear and free from organisms; just before death, twelve hours after the first spinal puncture, a second puncture showed fluid loaded with meningococci. This is the natural sequence of the infection, but is seldom caught by laboratory examination.

4. It is obvious that a patient may die with meningococcus infection without showing symptoms of meningitis, and one who waits for the classical symptoms of meningeal involvement, before suspecting meningitis, may easily miss the diagnosis.

UNDULANT FEVER, — AGAIN

The frequency of this infection appears to be in direct ratio to the vigilance of the physicians in suspecting it and having the proper examinations made. Within one week in Phoenix, the infection was found in a prominent state official and in one of the leading physicians, neither of whom, so far as can be ascertained, has been exposed to infection through goat milk. It is safe to say that there are more cases of undulant fever in one year in Maricopa County than there are cases of typhoid fever. Except for the fact that the former is seldom fatal, it would be a public health problem of the first magnitude. It should even now engage the attention of our health officials, in an effort to find the best means of protecting the milk-drinking public.

FRANK ELLIS BIRD

Dr. Frank Ellis Bird, of Albuquerque, N. M., died in that city on July 7th, at the age of sixty-three. The cause of death was apoplexy. Dr. Bird was born in Illinois in 1866, and graduated from the Ohio Medical University in 1903. He came to New Mexico in 1911. Before moving to Albuquerque he was located in Cuba, N. M.

ROSCOE G. BAZELL

Dr. Roscoe G. Bazell, of Winslow, Ariz., a practitioner for twenty-three years in northern Arizona, died at Winslow on April 22nd, He was a graduate of the Medical College of Cincinnati, class of 1901, and located in Arizona in 1906.

WILLIAM LOWE

Dr. William Lowe, formerly of Elizabeth, N. J., died in Phoenix, on February 7th, of cancer of the kidney. Dr. Lowe came to Arizona about eighteen months ago for change of climate. He was located at the Grand Canyon Bridge as emergency physician during the construction of that work. He was fifty-six years of age, and a graduate of the Medical Department of the University of New York, class of 1898.

PERSONALS AND NEWS

DR. GEORGE SHIELDS, of Phoenix, has returned to his practice after absence of a month, during which time he attended the Portland meeting of the American Medical Association and engaged in special work in urology.

DR. KIMBALL BANNISTER, of Phoenix, left August first for a month's postgraduate work in surgery in the Chicago clinics.

DR. R. B. RANEY, of Phoenix, has returned after absence of two months and has resumed his practice.

DR. DUDLEY FOURNIER, of Phoenix, left August first for motor trip through the northwest and Canada; he will be gone a month.

DR. H. M. PURCELL, of Phoenix, returned to his practice the middle of July, after postgraduate work in his specialty of urology.

DR. HARLEY YANDELL, of Phoenix, took his postgraduate work at Barnes Hospital, St. Louis, during June and July, in the eye, ear, nose and throat clinics. He reports that Barnes Hospital will soon be the second largest hospital in the United States.

DR. FRED B.SHARPE, of Phoenix, left the latter part of July for Portland, where he will engage in postgraduate work for a month.

DR. HOWELL RANDOLPH, of Phoenix, motored with Dr. Sharpe to Portland, the latter part of July; after a short vacation, he will return to Phoenix.

DR. L. H. THAYER, of Phoenix, returned to practice on August first, after a month at Mormon Lake in northern Arizona.

DR. JOHN WIX THOMAS, of Phoenix, returned on July 31st from a six week's vacation in the middle west; he expects to be away the last two weeks in August, spending this time in southern California.

DR. T. E. McCALL, of Phoenix, is back at his office, after several weeks invalidism following operation for acute appendicitis.

DR. EDGAR H. BROWN, of Phoenix, is spending several weeks in post-graduate work in orthopedics, in Los Angeles and San Francisco.

DR. R. J. STROUD, of Tempe, State Superintendent of Health, attended the American Medical Association meeting in Portland, the middle of July, thereafter visiting clinics before returning to his duties.

DR. H. J. FELCH, Phoenix, city health officer, after several weeks on the coast, has returned to his office and practice.

DR. A. C. KINGSLEY, of Phoenix, is studying in the postgraduate schools and hospitals of New York City, in industrial surgery and medicine, for two months.

DR. H. P. MILLS, and wife, of Phoenix, will return the latter part of August from a tour of Alaska covering the greater portion of six weeks.

FELLOWSHIP IN RADIOLOGY IN THE CHARITY HOSPITAL OF NEW ORLEANS

Dr. Amedee Granger Director of the X-ray Department of the Charity Hospital announces that the Board of Administrators of that Institution has established a Fellowship in Radiology.

The Fellows are members of the House Staff, ranking between the Internes and the House Physicians and Surgeons, and at the completion of their year's work will receive a certificate of Fellowship and become eligible for the position of Assistant Radiologist.

Here is a splendid opportunity for graduates of Class A-1 Medical Colleges to obtain thorough training in one of the newest and most important Medical Specialties. The Hospital is one of the largest in this country and the number and variety of clinical material is abundant as shown by the fact that more than 35,000 radiographs are made annually.

Potassium Permanganate in the Treatment of Pneumonia. John L. Chester. M. D., Detroit, Mich., Annals of Int. Med., May, 1929.

The British Medical Journal of March 7th. 1925, reported the first known cases of pneumonia successfully treated by Potassium Permanganate, Dr. Herbert W. Nott of Birkenhead, England, having been responsible for the therapeutic experiment. The patients received standard solutions of the drug as retained enemas, recovered promptly, and convalesced more satisfactorily than by more orthodox methods. Later, Dr. Nelson J. Roche of Southsea. England, obtained equally satisfactory results with similar injections, which he reported in the same publication on March 12th. 1927.

Becoming cognizant of the original experiments. Dr. Chester's first case was an advanced and seemingly hopeless one of flu-pneumonia of tuberculous origin, which, having failed to respond to other methods of treatment, was given 4 oz. of a standard solution of Potassium Permanganate every 3 hours rectally, at Providence Hospital. The patient recovered with more celerity and less fatiguing effort than is customary in such circumstances. Through the co-operation of various Detroit physicians, (names given), an additional 23 cases of lobar and broncho pneumonia were thereafter subjected to the same treatment at Providence Hospital, with consistently good results, and but two deaths.

Under very adverse conditions at Eloise Hospital. through the courtesy of the superintendent and his staff, 20 cases of pneumonia were selected at random. All were severe and complicated, some with the chronic heart conditions of long standing, and nearly all admitted chronic alcoholics. Ten of these cases were handled by other than the Potassium Permanganate method. and all died. The remaining 10 received appropriate doses of the standard solution of the drug, with the result that there were 50 per cent recoveries in what was considered well-nigh hopeless circumstances.

Dr. Chester provides concise case histories in his paper, with detailed progress notes in each of the institutional cases. In addition, he reviews the chemical action of the drug, explains its method of preparation and administration, and intimates its possibilities in other diseases.

GOOD SAMARITAN HOSPITAL.

(Continued from July issue)

Report of case of Triplets. These triplets were delivered by Dr. Sharpe in the Good Samaritan Hospital on Thanksgiving, Nov. 29, 1928. They were

about six and one-half to seven months infants, one boy and two girls. The first baby was a breach delivery; the other two were delivered by version. They weighed three pounds two ounces, three pounds three ounces, and three pounds one ounce respectively. Baby No.2, male, was cyanotic from birth; it never cried, but it moaned at intervals. His pulse was weak, respiration shallow and irregular, and he died the same evening. This baby, no doubt, had a large intracranial hemorrhage. Baby No. 3 had frequent cyanotic attacks for the first four days, than commenced to improve slowly. Breast milk and six per cent karo solution were given by mouth in small amounts most of which was regurgitated. Breast milk was inadequate and on the seventh day lactic acid milk was given; weight then was lowest ——two pounds nine ounces. By the 31st day she had regained her birth weight and seemed to be doing well. On the 51st day, she had a severe cyanotic attack and died suddenly. Oxygen and cardiac stimulants were given without any improvement of the symptoms. Baby No. 1 seemed the strongest. She was able to cry and seemed bright. She took her food better, but had a severe cyanotic attack on the fifth day. Her lowest weight on the seventh day was two pounds eleven and one-half ounces and she regained her birth weight on the 19th day. She did well and gained satisfactorily until the 51st day when she had a severe cyanotic attack; pulse was 180, almost imperceptible. Oxygen was given. She had other attacks of cyanosis on the 52d, 67th, 71st, 74th, 75th, and 76th; on the 82d day she had a severe attack from which she did not rally. The baby weighed five pounds and five ounces at time of death and seemed to be doing well. No necropsies were obtained. Baby No. 1 and baby No. 3 lived 51 and 82 days respectively. Both infants had loud rough systolic murmurs heard all over the chests. Their pulse rate was uniformly fast at all times, ranging from 140 to 180 beats per minute. Both of these infants digested their food well. The bowel movements were normal one to two times a day.

We believe that each baby suffered from an intracranial hemorrhage as it has been proven that the cyanotic attacks are caused by previous intracranial hemorrhages.

DR. L. H. THAYER reporting the following case: The patient has had two previous pregnancies. The first one was premature and the second one was normal time. The patient is below normal mentally and is thin and apparently undernourished. Weight 107. Blood pressure, 110/60. The urine has been normal during her pregnancy. There is no definitely abnormal condition.

About the eighth month of her pregnancy the membranes ruptured and four hours later the baby

Kansas City Annual Fall Clinical Conference of the Kansas City Southwest Clinical Society

October 7, 8, 9, 10, 11, 1929, Kansas City, Missouri

Headquarters and Meetings at President Hotel.

OPERATIVE AND DIAGNOSTIC CLINICS daily from 8:30 to 11:30 at Allied Hospitals.

A COMPLETE POST-GRADUATE COURSE at the President Hotel. Twenty Classes each morning.

CLINICS AND CLINICAL LECTURES each afternoon by the following distinguished guests:

Dr. Chevalier Jackson, Philadelphia, demonstrating the uses of the bronchoscope and the removal of foreign bodies from the bronchi of the lungs. He will also give an address on "Pulmonary Congestions."

Dr. George W. Crile, Cleveland, will give an address on "The Surgical Abdomen" and will hold a surgical diagnostic clinic.

Dr. Thomas McCrae, Philadelphia, giving a clinic on "Unusual and Usual Medical Cases." He will give an address on "Differential Diagnosis of Certain Chest Lesions."

Dr. Bela Schick, Austria and New York City, will demonstrate his famous "Schick Test", give a clinic and an address on "Feeding Problems in Children."

Dr. William Allen Pusey, Chicago, will hold a clinic on "The Diagnosis and Treatment of Certain Skin Diseases." The subject of his address will be "The Use of Helio-therapy in the Treatment of Certain Skin Lesions."

Dr. Robert Osgood, Boston, will give an address on "Newer Methods in Treatment of Arthritis" and a clinic on "Polyarthritis."

Dr. J. C. Litzenberg, University of Minnesota, will give an account of his latest research on "Tubal Pregnancy," and will hold a clinic on "The Differential Diagnosis of Tubal Pregnancy."

Dr. Vilray P. Blair, St. Louis, will give a clinic and demonstration of "Plastic Work on the Face," and an address on "Newer Methods of Skin Grafting."

Dr. Andre Crotti, Austria and Columbus, Ohio, will give an address on the results of his research work in endemic goitre and will hold a goiter clinic.

Senator Henry J. Allen, Wichita, Kansas, will address a Public Meeting Monday Evening.

Dr. Joseph A. Capps, Chicago, Illinois, Diagnosis of Internal Medical Cases.

ENTERTAINMENT:

Round Table luncheons each day.
Public Meeting, Monday Evening.
Get-Together Smoker, Tuesday Evening.
Alumnae Dinners, Wednesday Evening.
Golf Tournament, Friday Afternoon.
Golf Dinner, Friday Evening.

weighing five pounds 12 ounces was born. The infant appeared to be fairly well developed but had a rudimentary penis; it could urinate. The baby was born at 12:30 a. m. Respiration appeared to be alright but about six hours later the baby became cyanotic and stopped breathing. Oxygen and artificial respiration were used and he again appeared alright. The heart sounds were good. The respiratory sounds however were bronchial and shallow. About two o'clock in the afternoon he became cyanotic again; then there was considerable difficulty in restoring respiration. At 4 he became slightly cyanotic but in general seemed to be much better and during the night appeared to be alright. During the next day there were occasional periods when he was cyanotic. About 11.00 he stopped breathing and the nurse was unable to resuscitate him.

In this case we had an undernourished mother and premature rupture of the membranes and premature birth and an infant weighing not much below the weight of her previous children.

Dr. Berger said that nephritis and syphilis are both important factors in causing premature deliveries. He said the most important thing in the treatment of prematures is the regulation of the heat so as to keep the babies warm. The food at first should not be too rich. Start with what the baby can take then increase the caloric value gradually. Many of these children are lazy feeders and need urging to eat enough.

Dr. Shields ask about spinal punctures in these cases.

Dr. Jordon said that in these cases the temperature varied quickly from 96 to 101 degrees and it was difficult to keep the heart regulated properly. in a large series of cases in which there was syanosis and death hemorrhage has always been found. Syphilis has been found not to be so important in the etiology of premature delivery as was formally thought. Nephritis is an important cause. Spinal punctures were not done on these cases.

Dr. Sharp said that in this case polyhydramnios was an important cause of the premature delivery. The first baby to die certainly had intracranial hemorrhages. The first baby delivered was the one that lived the longest. It was delivered naturally. The other two had to be helped—being locked together.

DR. F. B. SHARP discussed a Craniotomy of a dead child as follows: Craniotomy of the dead child was selected as the operation of choice to effect delivery in this case. It was indicated because of a forty-two hour labor in a primipara—the membranes were ruptured approximately that long—an old infected cervix, several vaginal examinations without special antispetic precautions, a contracted

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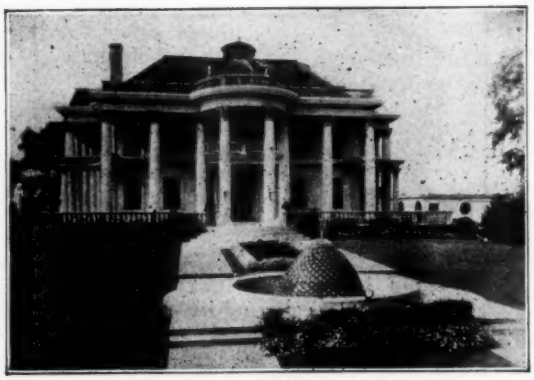

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pelvis, a large, dead baby, and poor general condition of the mother.

She was first seen in consultation with her physician thirty-six hours after labor had started. She was just awakening from an hour of ether anesthesia which had been given during an unsuccessful attempt to deliver the baby with forceps. Her pulse was 140, and of only fair volume. Considerable dehydration was present and the patient seemed on the verge of exhaustion.

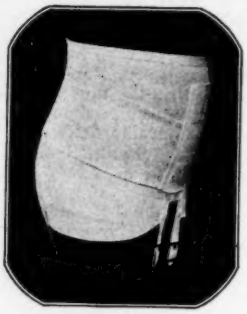
Further examination showed the uterus to be retracted closely around a fetus estimated to weigh nine pounds. The position was O. L. P., and the head was high above the inlet to the pelvis with much overriding of the pubis. Vaginal examination disclosed a pelvis markedly contracted in its internal dimensions, the outlet narrow, the sacrum convex laterally, and the true conjugate estimated to be about seven and one-half centimeters. The inlet was entirely filled by the occiput of the presenting head. There was much overlapping of the cranial bones and a moderate caput had formed. The cervix was completely dilated, but thick, edematous and eroded.

It was clearly evident that there was no possibility of the head ever coming intact through that pelvis. Fetal heart tones were present but rapid and irregular.

It was felt that the opportune time for a cesarean section had passed because of the time since rupture of the membranes, the vaginal examinations, the infected cervix with discharge, and the four applications of the forceps in an attempt to deliver the baby. The fetal heart tones gave evidence of approaching death of the baby which would certainly not be altered by abdominal delivery and its great risk to the mother. Accordingly, the patient was returned to bed and supportive treatment instituted. Pains began again and were severe. The fetal heart tones stopped within an hour. No progress in the labor resulted and as soon as it was felt that the patient could stand another anesthetic, preparations were made to do a craniotomy. The operation was begun about six hours after she was first seen. Complete collapse of the head was necessary in order to even bring it through the inlet of the pelvis. Cleidotomy and the use of the hook in the axilla were necessary to deliver the shoulders. A surprising amount of traction was needed to deliver even the hips. The placenta was then delivered manually because of atony of the uterus, and the uterus packed with gauze soaked in mercurochrome. A small transverse tear in the right vaginal wall was closed with catgut.

The patient suffered from a slight amount of shock but made a comparatively uneventful re-

"STORM"



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covery. She remained in the hospital eighteen days, and for a few days ran an afternoon temperature as high as 101.6. There was considerable pulmonary reaction to the anesthetic which contributed to most of the temperature.

This patient did not report to her physician for prenatal care until late in pregnancy. There was probably no opportunity for her physician to treat the infected cervix. The membranes were ruptured and she had been in labor for some time when she entered the hospital. Given a patient more co-operative, and with an understanding of the potential dangers of pregnancy, there would probably have been a better opportunity to have measured the pelvis carefully, estimated the relative size of the fetus, and upon noting the findings present to have done an early casarean section. Even after a trial labor of several hours a section is relatively safe if the low cervical type is done. It is hard to claim any safety, however, for any abdominal delivery done under the conditions in this case.

DR. J. M. GREER said that he knew nothing of this case except what he had heard this evening. He referred to the question of antenatal care. He thinks the public needs a great deal of education so they will appreciate proper prenatal care. He implored the doctors to do what they can to educate their patients on the importance of prenatal supervision.

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A TWO-CAR FAMILY

A two-car Springfield family who own their own home have three children. The oldest, a boy, flunked out in one high school course; the second a girl of ten, passed third grade work this time after spending a second term in that grade; the youngest, a boy going on seven, must spend another year in

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the first grade. This record was disclosed on report cards a month ago when school closed for the summer.

None of the children is "dumb." Their difficulty in school is almost entirely a health problem and not very complicated at that. All three have to shift for themselves at breakfast because evening social activities keep the mother up too late for rising before nine; the father deals in real estate and sleeps late, while the maid takes advantage of the situation and does nothing important about the morning meal.

Indulging their appetite for candy and soft drinks is not seriously limited by anything but their physical capacity and bedtime is a matter largely determined by the whims of the children.

On paper this sounds bad and extraordinary but there must be a lot of children in much the same circumstances. This is a moderately well-to-do family who pass as good respectable people of rather more than average intelligence. The parents simply haven't studied the problem of child raising sufficiently to realize the importance of habit and the regulation of diet.

This is a concrete illustration of the very active need for more general health education. People who do public health work are apt to get the notion that everybody knows about the health problems which seem primary to the worker.

Family situations like the one described are really of no little public concern. It takes from $75 to $120 per pupil to pay the annual operating expenses of the schools. Furthermore, when children grow up to become non-producers or partial producers the public pays the difference. When the reason is an easily preventable health problem the matter of conducting health education campaigns seems to be a plain public responsibility. (Illinois Health Messenger).

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